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A PSYCHO-BIOLOGICAL STUDY OF A SET OF IDENTICAL GIRL TRIPLETS

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The phenomenon of human multiple births has always attracted the attention of those engaged in the observation and study of the various facets of human biology. Twins, in particular, have been the subject of numerous reports, and the literature at present includes well over two thousand papers dealing with various aspects of twins and twin births. During the last decade, research workers in the fields of genetics, medicine, psychology, and education have turned their attention to the quantitative study of twins as constituting material which has unique possibilities for elucidating the relative effectiveness of heredity and environment on the physical and mental characteristics of the human being. The work of such men as Dahlberg (13), Verschuer (52), Newman (31, 32, 33, 34, 35, 36), Smith (50), Gesell (19), Bauer (6), Goldberg (21), Rosanoff (40), Wilson (60), Jones (60), Fisher (16), Siemens (47, 48, 49), Stocks (51), and others gives evidence of considerable progress in devising techniques for pursuing this fascinating study. So far, the study of triplets has been quite generally neglected, probably because of the relatively small number of triplets available.[1]

It is usually agreed at the present time that twins may develop as either one or two zygotes, the so-called identical and fraternal twins. In either type of twin, the uterine environment is probably more nearly alike for the individuals concerned than for ordinary siblings, but for identical twins the hereditary make-up is also identical, which fact accounts for the unique importance of this material for biological research purposes. The evidence reported by Arey (2, 3, 4), and Newman (33), as well as other evidence, leaves but little room for doubting the one-egg origin of identical twins. However, as far as the writer is aware, similar evidence of possible one-egg origin in the case of triplets is lack-

[1] Knibbs (23), in an analysis of the births occurring in Australia from 1890 to 1925, found that out of each million maternities 10,218 were twin births, 94.5 were triplets, and 1.5 quadruplets.

ing. Theoretically, triplets may develop from one, two, or three eggs, giving rise to monozygotic, dizygotic and trizygotic triplets. Sasse (43) has reported the most extensive study of so-called one-egg triplets. He gives full descriptions of two such cases of his own and, in addition, reviews thirty-two cases taken from the literature. In twenty-seven of the thirty-four cases but one chorion [2] was present. Of the twenty-seven monochorionic cases, three amnions were reported in nineteen, two amnions in five, and one single amnion in three cases. Sasse concludes: "Eineiige Zwillinge sind keine gar zu seltene Erscheinung. Drillinge dieser Art kommen dagegen recht spärlich vor." Williams (59) reports detailed analyses of the placental organization found in six triplet births, concluding that three of the cases were dizygotic, two trizygotic, while in one case it seemed possible that the triplets were monozygotic. Randall (39), Waldner (54), Suzor (51a) and many other practitioners have reported obstetrical details of triplet births, usually with insufficient detail to be of much value to the investigator interested in the comparative study of triplets. Many of these papers deal primarily with the difficulties connected with triplet deliveries. It is the general conclusion of the various workers that dizygotic triplets are the most common, while trizygotic triplets are quite rare, and monozygotic triplets very rare. One recent triplet study deserves special mention. Komai and Fukuoku (25) report a case of triplets for which a rather complete set of comparisons were made, including finger, palm and sole prints, certain physical measurements, and a number of psychological tests. The results of these comparisons lead the authors to conclude that the triplets are monozygotic, although a careful analysis of the preserved placenta reveals the presence of two chorions. The latter finding would tend to cast some doubt on the one-egg origin of the triplets, although it is known that in a small number of cases the placental evidence may be misleading.

Detailed studies of triplets in which the members are carefully compared by using psychological and physical measurements are but few. Fisher (17) has reported the most extensive study of triplets to date. He secured his cases through the records of the Royal Bounty which is granted by the British government to surviving triplets. He made a biometrical analysis of six body dimensions taken when the triplets were six and one-half years old, as well as a study of mortality findings.

[2] The presence of but one chorion is usually taken as evidence of monozygotic origin.

His study is outstanding because of the comparatively large number of cases, care in selection of population studied, and the careful and exact use of biometrics in the analysis. His conclusion, that about fifty-four per cent of like sexed triplets are monozygotic is of particular interest. Rowe (41) gives a very incomplete report dealing with a set of triplets consisting of two very similar girls and a boy. Butts (9) reports a psychological survey of a set of six-year old girl triplets, concluding them to be trizygotic. Clark and Revell (10) report a fairly comprehensive study of two sets of very similar triplets, making use of finger and palm prints, certain measurements of physical dimensions, as well as making a report of the developmental history. Gates (18) has published a paper dealing with one of the sets of triplets studied by Clark and Revell, in which he points out the necessity of using the triplets' siblings as control data, particularly with reference to the analysis of facial and general physical similarity. Two additional triplet studies deserve mention. Newell (30a) gives a rather complete description of a pair of surviving individuals from a triplet birth. This case is of particular interest because of the pronounced mental and physical differences of the surviving individuals despite the fact that indications are that they are one-egg twins. The difference is ascribed to intrauterine differences and the effects of disease in the case of one of the individuals. Sanders (42a) presents rather complete data on two sets of triplets, paying particular attention to anthropometric measurements, frictionridge comparisons, intelligence tests, and developmental histories. Both sets are presumably monozygotic.

HISTORY OF THE TRIPLETS

The present paper reports the findings of a rather intensive study of a set of ten year old girl triplets, together with certain control data for the six remaining siblings and the parents.[2] The Johnson family (Plate

[2] Certain comparisons to be reported were made possible by a grant of money from the Graduate School Research Fund of the University of Minnesota. In addition the writer gratefully acknowledges the interest, coöperation, and professional help of the various members of the faculty and staff of the University Hospital, School of Dentistry, Institute of Child Welfare, and the College of Education, to whom specific acknowledgment will be made at various points in this paper. The writer also acknowledges his debt to Mr. and Mrs. J. N. Johnson for permission to bring the triplets to Minneapolis for the examination, and to Esther, Ethel and Esmer who were so patient and coöperative throughout, as well as to Alice, who assisted the writer in securing the family history, photos, etc.

I) is of Swedish descent, consisting of seven girls and two boys. All of the siblings have been reared on the farm. The environment as far as home, nutrition, parental care, etc., is concerned, can be considered as being average. The triplets were born December 7, 1921 at the Boysen hospital in Pelican Rapids, Minnesota. The triplets resulted from the sixth of the seven births in the family, which is in accord with the finding of various investigators that multiple births are more frequent for the higher age ranges of the mothers' period of fertility (Dahlberg, 14). There is no history of multiple births on either the paternal or maternal side. At the time of the triplet birth the father was 39 years old and the mother 38, while the age range for the siblings is 16 years.

During the triplet pregnancy the mother's health was poor, due to a rather severe mitral stenosis, and a pronounced toxemia probably due to renal insufficiency. During the last period of pregnancy the mother was very edematous and had to take to the bed. The delivery occurred after a term of approximately eight months, was not particularly protracted, and offered no serious difficulties. The interval between the birth of each triplet was approximately twenty minutes. The birth order was Esther (X) three pounds fourteen ounces, Ethel (Y) three pounds fourteen ounces, and Esmer (Z) four pounds one ounce.[4] The neo-natal condition of the triplets was very poor, Y in particular being markedly cyanosed and very weak, and it was largely the heroic efforts of the nurse in charge which accounted for Y's survival. Dr. P. Boysen who attended the birth was unable to give the writer exact information with respect to the organization of the placenta, outside of the fact that it was single and of approximately normal size, with the umbilical cords inserted in three separate places but quite close together. Although a single placenta has some significance for diagnosing the type of twins and triplets, it is impossible to infer that a single chorion also obtained, for two or more placentae will often fuse and present the appearance of only one (Williams, 59). The first year of life was quite uneventful, although Z had a slight attack of convulsions, and Y made slow progress to begin with. Fortunately, the mother kept a rather complete weight record (Table 1), during the first year. A glance at Figure 1 indicates that Y was slower in developing throughout the first year; but, despite Y's lower weight, the parallelism in weight increment is striking.

The developmental history of the triplets is fragmentary, being based largely on the memory of the mother and the oldest sister Alice. In

[4] Hereafter the triplets will be referred to as X, Y, and Z.

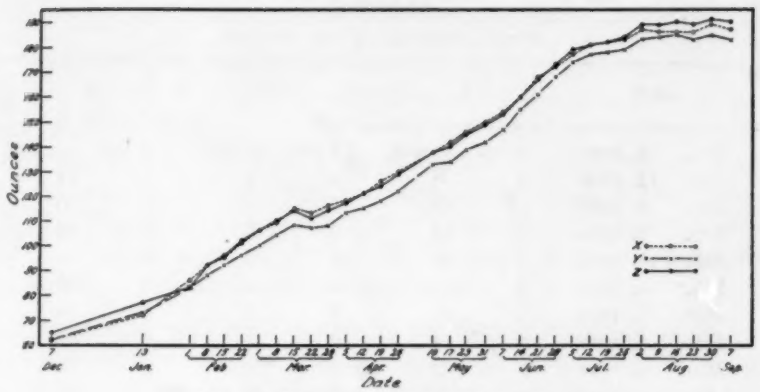

FIG. 1. WEIGHT RECORD OF TRIPLETS

general, the development was very similar and parallel throughout the years of early childhood. (Plates II and III). The triplets crept very little. Shortly after the seventh month the form of locomotion preferred was that of walking on all fours. Actual walking occurred at approximately one year. The development of speech was very slow, although the triplets seemed to communicate with each other very early. Real speech was established at about three years, which is in accord with the findings of Day (15) who observed that the development of speech in twins is as a rule, retarded. The triplets had their own names for each other: "Kakibee" (X), "Keevie" (Y), and "I Am" (Z). The children were very active, enjoyed playing with each other, and rarely quarrelled. Tooth eruption was very nearly the same, the mother indicating that usually there was not more than a day's difference in the time of eruption of a certain tooth (?). Such childhood diseases as were experienced, were usually shared together, although Z was most often the one who became ill first. As we shall see later, this close parallelism does not necessarily indicate that the anatomical and physiological effects were the same for all three. All three had measles when five years old with very similar progress of the disease. They were vaccinated for smallpox shortly after this, with positive results. The triplets have always been very similar with respect to physical likeness, similarity of responses, likes, dislikes, and general attitude. During the first few years of life it was difficult to identify them, and consequently they were "tagged"; for even the mother would make mistakes. Interestingly enough, the younger brother John never had any difficulty in identifying the triplets. Even at this late day, the writer was interested

TABLE 1

Weight Records of the Triplets

DATE	X	Y	Z
Dec. 7, 1921	3 lbs. 14 ozs.	3 lbs. 14 ozs.	4 lbs. 1 oz.
Jan. 13, 1922	4 8	4 9	4 13
Feb. 1, 1922	5 6	5 3	5 6
Feb. 8, 1922	5 12	5 8	5 12
Feb. 15, 1922	5 15	5 12	6 0
Feb. 22, 1922	6 5	6 0	6 6
Mar. 1, 1922	6 10	6 4	6 10
Mar. 8, 1922	6 13	6 8	6 14
Mar. 15, 1922	7 3	6 12	7 2
Mar. 22, 1922	7 1	6 11	6 15
Mar. 29, 1922	7 4	6 12	7 2
Apr. 5, 1922	7 6	7 1	7 5
Apr. 12, 1922	7 9	7 3	7 9
Apr. 19, 1922	7 14	7 6	7 12
Apr. 26, 1922	8 2	7 10	8 1
May 10, 1922	8 10	8 5	8 10
May 17, 1922	8 14	8 6	8 12
May 23, 1922	9 2	8 11	9 1
May 31, 1922	9 6	8 14	9 5
June 7, 1922	9 10	9 3	9 9
June 14, 1922	10 0	9 11	10 0
June 21, 1922	10 8	10 1	10 7
June 28, 1922	10 12	10 8	10 13
July 5, 1922	11 1	10 14	11 3
July 12, 1922	11 5	11 1	11 5
July 19, 1922	11 6	11 2	11 6
July 26, 1922	11 7	11 3	11 8
Aug. 2, 1922	11 11	11 7	11 13
Aug. 9, 1922	11 10	11 8	11 13
Aug. 16, 1922	11 10	11 9	11 14
Aug. 23, 1922	11 10	11 7	11 13
Aug. 30, 1922	11 13	11 9	11 15
Sept. 7, 1922	11 11	11 7	11 14
Oct. 11, 1922	11 13	11 13	11 15
Nov. 1, 1922	12 2	11 15	12 8
Nov. 1, 1925	28 8	30 0	28 8
Oct. 1931	47 5	48 3	45 6

in observing that the mother was not sure which one of the triplets lay sleeping in a bed some twenty feet distant, although the child was plainly visible.

LIKENESSES AND DIFFERENCES

With respect to certain differences, the writer noted that when folding their hands, X places the right thumb over the left, while Y and Z place the left over the right. All use the right hand for writing, catching a ball, etc., and the right leg for kicking; but, in crouching for a quick start in running, Y leads with the right, while X and Z lead with the left leg. The writer was unable to secure consistent results for eyedness. For direction of whorl of the hair on the head, X has a counterclockwise, while Y and Z have clockwise whorls.

During their visit to Minneapolis, the triplets stayed in the home of the writer, and he was, therefore, together with Mrs. Lund, accorded an invaluable opportunity to observe the every day activities of the triplets, both when well and during the illness of all three. The writer readily learned to identify them when all three were present, and, by being careful, when they appeared singly. In general personality characteristics the triplets are very much alike, although there are some differences. X is somewhat more quiet and is an avid reader. She is also the most patient when ill. Z is the most talkative, and appears to be somewhat more active than either Y or X. She is also patient when ill. Y is more impetuous and impatient, and was somewhat trying to care for when ill. However, the impression of the writer is that no two of the triplets are consistently the most alike.

An examination of the family photograph indicates that the triplets resemble each other more than either of them resemble any of the remaining siblings, and more than any two of the siblings resemble each other. Such difference as is noted is largely due to differences in facial expression or posture at the time, although there is a noticeable difference in the case of Esther X, whose habitual expression is somewhat different from that of Y and Z (Plate III). In certain genetic details there is quite a marked resemblance between the siblings, while in other details the differences are very marked. Samples of the hair of the triplets were submitted to several persons for examination and they all agreed that the hair was so similar in color and texture that it could have been taken as the hair of one person on three different places of the head. The hair of the siblings ranges in color from a very light blond, to jet black. The sibling range with respect to eye pigmentation is also very wide, as measured by the Saller chart (42) (Table 2).

TABLE 2

Eye Pigmentation and Approximate Hair Color of the Johnson Family

	SALLER CLASSIFICATION OF EYE PIGMENTATION	HAIR COLOR
Father	M 10	Jet Black
Mother	M 14	Medium Auburn
Alice	P 5	Black
Albert	P 4	Black
Edna	S 1	Medium Auburn
Lillian	M 2	Light Auburn
Anna	M 21	Very Light
Esther (X)	P 2	Black
Ethel (Y)	P 2	Black
Esmer (Z)	P 2	Black
John	M 14	Medium Auburn

FINGER PRINTS

The use of dermatoglyphics for diagnosing the type of twins has received a great deal of attention and, in general, the consensus of opinion seems to be that such comparisons furnish one of the best available clues in the absence of positive placental findings. Finger, palm and sole prints were therefore taken of the whole family (prints of the soles were taken for the triplets only).[5] Each print has been carefully studied and formulated by the writer. The friction skin of the triplets is very smooth in contrast to that of the remaining members of the family, and it was necessary to take four sets of prints before at least one good print was secured of each finger, and, in addition, each print was formulated three times independently as a check. The findings with respect to the finger prints are set forth in Table 3. Newman's method has been used in assigning numerical values to the finger ridge counts. Examination of the reproduction of the triplet's finger prints (Plate IV) convinces one of the striking similarity. When all three triplets are compared at once, the patterns on five of the ten fingers are practically identical.

[5] The prints were taken by means of a glass plate, lamp black ground in oil, and a printer's roller. Ordinary bond paper was used to receive the impressions. The methods used in formulating the prints are too involved and technical to be included in this paper, and the reader is therefore referred to papers by Wilder (57, 58), Newman (32, 33, 34), Cummins and others (11), Montgomery (30), Komai (24), and Stocks (51).

TABLE 3

Finger Print and Ridge Counts *

	PATTERNS					RIDGE COUNTS	TOTAL
Father	W^c	W	W	W^c	U	10-13-12-10-12	57
	W^c	W	W	W	U	14-12-11-13-12	62
Mother	W^l	U	U	U	U	11- 4- 6-10- 5	36
	U	U	U	U	U	11- 4- 7- 8- 5	35
Alice	W^c	W^{sp}	U	W^c	U	14-11- 9-13-12	59
	W^c	W	U^w	W	U^w	16-13-14-15-13	71
Albert	W^c	W^r	U	W	U	15-15- 9-13- 9	61
	U	U	U	U	U	16-17-19-14-12	63
Edna	W^{sp}	W	W	W^c	U^c	12-11- 9- 9-11	52
	W	W^c	W	W^c	U^w	16- 8-11-12-11	58
Lillian	W^c	W^{sp}	U	W	U	13- 8- 9-10- 8	48
	W	R	U	U	U	15-10-11-15- 9	60
Anna	W	A	U	W	U	12- 1- 7-14-13	47
	W^{sp}	R	U	U	U	15-13-11-14-12	65
Esther	U	W^a	U	W^l	U	14-11-10-13-11	59
	U	U	U	W^l	U	11- 8-12-11-12	54
Ethel	U	U	U	W^l	U	13- 9-11-12-11	56
	U	U	U	U^w	U	11- 6- 9-12-11	49
Esmer	U	U	U	W^l	U	13-11-13-14-13	64
	U	U	U	W^l	U	12- 9-14-13-12	60
John	W^c	R	U	W^c	U	8-12- 9-11- 9	59
	U	W	W	W^c	U	12- 9-12-10- 9	52

W—common whorl, W^c—composite type, W^{sp}—spiral type, W^r—related to radial loop, A—arch, W^l—transitional pattern of loop type, U^w—transitional pattern of whorl type, U—ulnar loop, R—radial loop.

* Formula for right hand is given first in each case.

When X and Y are compared 8 fingers are nearly identical; for X and Z, 7 fingers; and for Y and Z, also 7 fingers are alike. Some differences are noted. X has a whorl on digit 2 of the right hand, while on the corresponding finger Y and Z have loops; the whorl in the case of X is, however, a transitional type, clearly a loop as far as axis and location of triradius is concerned, but the presence of at least one clearly recurving line makes it necessary to classify it as a whorl. A similar finding holds true for the whorls that are present on digit 4 of all hands except the left of Y. All the whorls for digit four are of the transitional type, but for Y's left, the absence of a recurving line classifies the pattern as a loop. In none of the siblings do we find this transitional type of whorl.

The prints of the triplets resemble those of the mother with respect to pattern type and the father with respect to ridge count. All in all, the finger prints of the triplets are as nearly alike as those usually found in identical twins, and this close similarity alone is rather good evidence in favor of monozygosity.

PALM PRINTS

In formulating the palm prints the writer has made constant use of the very excellent paper by Cummins and others (11). In addition, the triplet prints were submitted to Dr. H. H. Newman who checked the formulation, suggesting a few slight changes in the right half of the formulae[6] (Table 4). Again, comparing palm prints, we find the triplets to be very similar (Plate V). For the D-line (first term in formula) we find the triplets and the siblings quite alike except for one hand of each for Lillian, Anna and John. For this line, the triplets are alike except for a reversal in the case of X. For the C-line (second character) the sibling resemblance is not as great, but the triplet resemblance is striking. Only in the case of the mother and two siblings do we find similar X-formulations. For the B-line, terminations similar to those of the triplets are found in five of the twelve sibling hands. X again shows a reversal. For the A-line, the triplet palms are nearly identical in the course the line takes to its termination. A similar termination is found in only three of the sibling hands. With the exception of the right hand of X, the axial triradius is found in almost exactly the same place in each of the hands. The outstanding difference in the triplet hands is found on the hypothenar area of X where a complete loop is found, while for the other triplets the patterns are ulnar arches. The triplets clearly resemble each other more than they do the siblings, and more than any two of the siblings resemble each other. Newman[7] characterizes the triplet palms as follows:

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[6] The reader who is unfamiliar with the scheme of formulation used is referred to the paper by Cummins and others cited above. Suffice it to say that the first four sets of numbers indicate the termination of each of the four main lines, one such line arising from each small triangle usually found at the base of each finger. The single character in the middle of the formula indicates the presence and location of additional triradii in the hand, while the last five characters indicate the presence and type of patterns found on the heel of the hand (hypothenar), at base of thumb (thenar) and the second, third and fourth interdigital spaces.

[7] Private communication.

All six palms are of a rather pronounced right-handed[8] type. Right hands of both X and Z are definitely more right-handed in type than are their respective lefts. Y shows slight asymmetry reversal, the left hand being slightly more right-handed than is the right. Left hands of Y and Z are nearly identical, being more similar than is either to her own right hand. The right hands of X and Z are more similar than is either right to own left. All of these interrelations are, I think, just what one might expect in monozygotic triplets.

TABLE 4

*Palmar Dermatoglyphics of the Johnson Family**

	D	C	B	A	Ax	H	T	1	2	3
Father	11(10)	. 7	. 7(6)	. 2	— o	— A^r	.	o	. M	. o . L
	11(10)	. o	. 7(6)	. 3	— t′	— A^u	.	o	. o	. L . M
Mother	11(10)	. 9	. 7(6)	. 5′	— t″	— L^c/L^uV	.	o	. M	. L . V
	11(10)	. X	. 5″(6)	. 3	— t″t	— L^c/L^u	.	o	. M	. V . V
Alice	11	. 9	. 7	. 2	— o	— A^r	.	o	. o	. L . V
	11(10)	. 9	. 7(6)	. 2	— o	— A^r	.	o	. M	. 1 . M
Albert	9(10)	. X	. 5″(6)	. 5′	— t′	— L^u	.	o	. M	. V . M
	9	. 7	. 5″	. 3	— t	— W^s	.	o	. M	. o . L
Edna	11	. 7	. 9	. 3^h	— t	— L^r	.	o	. o	. 1 . M
	11(10)	. 7	. 7(6)	. 3^h	— t	— L^r	.	o	. o	. o . L
Lillian	11	. 7	. 9	. 3^h	— o	— A^r	.	o	. o	. L . o
	9	. 7	. 5″	. 3^h	— o	— A^r	.	o	. M	. M . 1
Anna	11	. X	. 7	. 5′	— t′	— o/A^cV	.	o	. o	. V . M
	9(10)	. o	. 5″/6	. 5′	— t′	— V/A^c	.	o	. o	. o . M
Esther	11	. 9	. 7	. 5′	— tt″	— L^r/A^c	.	o	. V	. L . o
	11(10)	. X	. 7(6)	. 5′	— t	— A^u/A^c	.	o	. o	. V . o
Ethel	11(10)	. X	. 7(6)	. 5′	— t′	— A^u/A^c	.	o	. M	. V . o
	11	. X	. 7	. 5′	— t	— A^u/A^c	. V	. o	. V	. V
Esmer	11(10)	. 9	. 7(6)	. 5′	— t′	— A^u/A^c	.	o	. M	. L . o
	11	. X	. 7	. 5′	— t	— A^u/A^c	. V	. o	. o	. V
John	11(10)	. 9	. 7(6)	. 2	— t′	— o/A^cV	.	o	. o	. L . o
	9	. 7	. 5′	. 2	— t′	— o/A^cV	.	o	. o	. o . 1

* Formula for right hand is given first in each case.

SOLE PRINTS

The sole prints are particularly interesting, for here the similarity is extraordinary. The writer failed to find any method of formulation which would express in an approximate manner the patterns found, and has therefore formulated the sole prints in a manner analogous to that of the right-hand half of the palm prints, indicating merely the presence,

[8] See Newman (35).

type and direction of the 7 possible patterns (Wilder, 57). The formulations are presented in Table 5.

TABLE 5

Sole Patterns of the Johnson Family ‡

	HALLUCAL	SECOND INTERDIGITAL	THIRD	FOURTH	HYP.	CALC.	THENAR
Father	L^{td}*	o	o	o	$L^{t}V$	o	o
	L^{td}	$L^{p}V$	o	o	$L^{t}V$	o	o
Mother	W	o	(L^{d})		L^{t}	o	o
	W	$L^{p}V$	L^{d}	L^{d}	$L^{t}V$	o	o
Alice†							
Albert	L^{td}	o	(L^{d} — $L^{d}V$)		$L^{t}V$	o	o
	L^{td}	$L^{p}V$	(L^{d})		L^{t}	o	o
Edna	W	L^{p}	L^{d}	o	L^{t}	o	o
	W	$L^{p}V$	L^{d}	$L^{p}V$	L^{t}	o	o
Lillian	L^{d}	L^{p}	(L — $L^{d}V$)		$L^{t}V$	o	o
	L^{td}	$L^{p}V$	o	o	L^{t}	o	o?
Anna	W	o	(L^{d} — $L^{d}V$)		$L^{t}V$	o	o
	L^{t}	$L^{p}V$	(L^{d})		L^{t}	o	o
Esther	L^{t}	$L^{p}V$	L^{d}	L^{d}	L^{t}	o	o
	W	L^{p}	L^{d}	L^{d}	L^{t}	o	o
Ethel	W	$L^{p}V$	L^{d}	L^{d}	L^{t}	o	o
	W	L^{p}	(L^{d})		L^{t}	o	o
Esmer	W	$L^{p}V$	L^{d}	L^{d}	L^{t}	o	o
	W	L^{p}	L^{d}	L^{d}	L^{t}	o	o
John	L^{tp}	$L^{p}V$	L^{d}	$L^{d}V$	$L^{t}V$	o	o
	L^{tp}	$L^{p}V$	$L^{d}V$	o	$L^{t}V$	o	o

* W—whorl, L—loop, V—vestigial. Exponential letters indicate direction of opening of loop: t—toward tibial side, f—toward fibular side, p—toward heel end, d—toward toe end, tp and td indicate diagonal opening. ( ) two interdigital areas covered by one large pattern. (—) two patterns fused.

† No record.

‡ Formula for right sole is given first in each case.

Five of the hallucal whorls on the triplets' feet (Plate VI) are very similar while the sixth, right of X, is a loop of the transitional type, resembling a whorl more than a typical loop. In addition, the hallucal whorls of the triplets are unlike any of those found in the siblings or those of the mother, all of which are elliptical, while those of the

triplets are circular. No sibling has a hallucal pattern like the right of X. For the second and third interdigital areas, both feet of Edna and Lillian show a decided resemblance to the corresponding areas of the triplets. For the third and fourth areas Y has one large pattern covering both areas. Patterns similar to the triplets' hypothenar are found on five of the siblings' twelve feet. No calcar patterns were found, and only in the case of Lillian's left foot was there any indication of a pattern on the thenar area. Again we conclude that the triplet resemblance is closer than is the resemblance of any two siblings. Montgomery (30) states, ". . . . the presence of identical patterns on the soles of a pair of twins might point to their monozygotic origin, but as Newman states, their absence does not disprove it."

MEASUREMENTS

Anthropometrical measurements have been widely used in twin research, but until recently there has been no method of analyzing the results. The writer feels particularly fortunate in having secured the services of Dr. Edith Boyd of the Institute of Child Welfare for the measurement of the external bodily dimensions of the triplets. (Table 6) The measurements were made without clothes, using the standard instruments, and carefully controlling body posture. Boyd (8) has shown that each bodily measurement has its own experimental error of

TABLE 6

Measurements of External Body Dimensions of the Johnson Triplets

	X	Y	Z
Standing height **	131.2 Cms.	131.2 Cms.	127.6 Cms.
Stem length (knees up)	66.8	67.8	67.3
Sitting height (legs str.)	66.4	67.0	67.2
Sitting height (legs hanging)	67.4	69.0	67.6
Circumferences			
Fronto-occipital **	51.8	51.8	52.0
Supraorbito-occipital **	50.8	50.9	51.1
Vertex-mental	58.0	58.1	57.8
Neck	24.6	25.1	24.2
Bihumeral	67.8	67.6	66.9
At nipple (quiet)	54.5	57.0	54.5
At nipple (inspiration)	57.0	58.8	58.4
At nipple (expiration)	53.0	55.0	53.7
Apex costal angle	53.6	55.0	53.5

Tenth rib	49.9	52.9	49.4
Umbilical	48.7	47.0	47.2
Iliacal **	50.2	47.3	47.2
Trochanteric **	58.2	57.8	57.7
Thigh *	31.6	31.8	31.0
At knee *	24.4	24.2	23.8
At calf *	22.2	21.8	21.0
At ankle *	13.9	14.5	14.4
Foot (base little toe)	16.5	16.5	15.9
Axillary *	15.8	16.2	15.5
Maximum biceps *	14.4	15.2	14.5
Maximum forearm *	14.5	15.0	14.2
Minimum forearm *	10.7	10.8	10.4
Wrist	11.2	13.7	11.0
Anterio-posterior diameters			
Fronto-occipital **	18.0	18.5	18.2
Supraorbito-occipital **	17.4	17.7	17.7
At nipple	12.1	12.5	12.0
Apex costal angle	12.1	13.7	12.1
Tenth rib	13.1	13.5	12.3
Umbilical	12.9	12.6	11.2
Sacro-pubic	13.5	12.6	12.6
Transverse diameters			
Biparietal *	13.1	13.3	13.5
Bimalar	9.0	9.0	9.6
Bigonial *	8.7	8.7	9.5
Bicondylar *	10.1	10.4	11.1
Biacromial	24.5	24.0	24.0
Bihumeral	28.1	28.7	26.5
At nipple	18.4	18.7	17.9
Bipapillary	12.9	14.3	12.9
Apex costal angle	17.8	18.9	17.8
Tenth rib	17.7	18.0	16.1
Umbilical	16.8	16.1	16.9
Bisiliacal **	20.3	19.3	19.3
Intertrochanteric **	21.2	20.8	20.9
Interspinous *	19.6	17.5	18.7
At knee *	7.1	7.0	7.0
At ankle *	5.7	5.6	5.4
Of foot *	6.2	5.9	6.0
Elbow	4.9	5.0	4.9
Elbow	4.4	4.6	4.5
Wrist *	3.8	3.9	3.7
Metacarpophalangeal *	6.0	6.1	5.7

Actual lengths			
Vertex-menton	21.9	21.9	21.7
Nasion-menton *	10.4	10.2	9.7
Sterno-pubis	37.5	38.7	38.2
Sterno-angulus *	9.7	10.2	10.3
Sterno-umbilicus	26.0	25.2	26.7
Angulo-pubis	27.9	28.3	28.6
Angulo-umbilicus	16.7	15.2	16.9
Umbilico-pubis	10.9	12.8	11.4
Costo-iliaca	5.0	5.6	4.8
Iliaco-ischium	15.9	17.0	16.9
Iliaco-genu **	43.6	43.2	42.1
Trochantero-genu	37.7	34.5	34.4
Genu-malleolus **	32.2	31.9	31.2
Malleolo-planta	5.0	4.5	4.7
Of foot *	19.9	20.0	19.7
Acromio-stylion (straight)	41.2	41.9	41.0
Acromio-stylion (flexed)	...	...	...
Acromio-radiale	22.9	24.1	23.6
Radio-stylion **	18.9	18.5	17.9
Stylio-medius *	13.9	15.8	13.7
Medius *	6.0	6.0	5.8
Trochantero-planta	71.1	70.0	69.9
Midline lengths			
Vertex-planta	131.3	131.2	129.2
Vertex-nasion	11.7	11.7	11.0
Vertex-menton	20.0	20.0	19.9
Vertex-sternum	24.9	25.1	25.4
Vertex-angulus	34.0	34.7	34.7
Vertex-umbilicus	50.5	50.1	51.5
Vertex-pubis	61.6	62.7	63.1
Lateral lengths			
Vertex-meaton	11.9	12.1	11.5
Vertex-acromium	21.1	21.1	23.7
Vertex-iliaca	51.8	53.5	52.3
Vertex-medius	76.7	77.4	79.9
Vertex-stylion	63.2	63.5	66.7
Vertex-trochanter	58.5	61.5	60.7
Vertex-genu	93.9	95.2	94.6
Vertex-malleolus	125.6	126.6	125.0
Vertex-rump	70.7	70.5	70.1

measurement, and inasmuch as most of these experimental errors are of unknown magnitude it is difficult to evaluate differences which are found

in the measurement of twins and triplets. Ninety-three measurements were taken (Table 6). The writer has worked out a method of making quantitative comparisons of the degree of similarity of triplets with respect to the measurements of bodily dimensions. The method is based on work previously reported by Aebly (1), Dahlberg (13), and Verschuer (52). Briefly, the method consists of rearranging the triplet data in a manner analogous to that of three pairs of twins, thus X-Y, X-Z, and Y-Z. For each such set of measurements the following calculations are made: For each dimension measured, the deviation of either individual from the mean of that measurement is calculated. This deviation is then divided by the mean of the measurement thus converting the deviation into per cent of the mean. The median of the ninety-three per cent deviations is then calculated, together with some measure of range or dispersion—in this case the first and third quartiles. Similar calculations are made for each of the two remaining comparisons. In order to give greater weight to very large or very small deviations, the per cent deviation may be squared, the mean squared per cent deviation calculated and the square root extracted, thus securing a result essentially comparable to the standard deviation of a series of deviations. The results of this analysis are set forth in Table 7, giving the constants derived from the distributions of the X-Y, X-Z, and Y-Z per cent deviations.

TABLE 7

Measures of Similarity of Triplets' External Body Dimensions

CONSTANT	XY	XZ	YZ
93 dimensions			
Third Quartile	2.10%	2.54%	2.54%
Median	1.09	1.12	1.31
First Quartile	.41	.53	.50
* *and* ** *dimensions* (34)			
Third Quartile	2.12	2.67	2.30
Median	1.01	1.33	1.37
First Quartile	.38	.67	.67
** *dimensions* (11)			
Third Quartile	1.30	2.56	1.30
Median	.87	.87	.87
First Quartile	.20	.42	.15

An examination of the constants in Table 7 discloses the fact that, although for the ninety-three dimensions the mean per cent deviation is slightly less for the X-Y comparisons, the dispersion is practically the same for all three comparisons, and that, therefore, the differences are probably not significant. Dr. Boyd selected 34 dimensions which, in her opinion, were somewhat more reliable than the average measurement, and 11 which she considered to be the most reliable. Separate calculations were made for these two series of measurements, with results which again demonstrate the essential similarity of the triplets. It is unfortunate that comprehensive norms are not available for the per cent deviations of same-sexed siblings, measured at comparable ages, although Dr. Verschuer has published tentative norms for identical and fraternal twins (52), as has also Dahlberg (13). In both cases the age range of the material is fairly wide, and, in addition, Dahlberg's norms have to be halved in order to be comparable to those of Verschuer. However, it seems safe to conclude that the similarity of the triplets is great enough to place them within the range of difference ordinarily found in identical twins.

Through the kindness of Dr. John Anderson of the Institute of Child Welfare, the Institute's photographer, Mr. D. Lawrence, made a photographic analysis of the triplets. Anterior, posterior and lateral views were taken of their nude bodies, in addition to shadow pictures to show postural attitude. No significant differences were found. Pictures were also taken of the triplets' hands, two views being taken of each hand. An analysis of the last named pictures discloses the fact that the triplet's hands are practically identical as to shape, finger lengths, and nail formation, which furnishes excellent corroborative evidence of their monozygosity.

X-RAY ANALYSIS

The writer is indebted to Dr. Leo Rigler, Head of the X-ray department of the University Hospital and to Mrs. O. S. Dahl, X-ray technician, for the very thorough and complete X-ray analysis of the triplets. Films were made of the hands, wrists, elbows, shoulders, feet, ankles, knees, pelves, lumbar spines, sinuses, mastoids, and the sella turcicas. The complete review of these findings is too extensive to reproduce in this paper and a short summary is given, paraphrasing from the report of Dr. Rigler who read and interpreted the films. In general, the results of the X-ray analysis show a greater degree of

similarity than any other comparison made, and it is the writer's opinion that the use of the X-ray in twin and triplet research has much promise.

Seven foot dorso-ventral teleoroentgengrams were made of the triplets' hearts (Table 8). There is a slight difference in the heart size, that

TABLE 8

Heart Measurement of Triplets Compared with Lincoln Norms for Appropriate Age and Sex

DIMENSION	LINCOLN NORM Mean	S. D.	X	Y	Z
Trans. thorac.			18.5 Cms.	18.8 Cms.	18.5 Cms.
Median-left	6.3	± .73	6.3	5.5	5.7
Median-right	3.2	± .54	3.2	3.5	3.5
Total	9.5	± .82	9.5	9.0	9.2
Longitudinal			11.0	10.5	10.3
Arch			2.0	1.8	2.0

of Y being slightly smaller than Z's which in turn is slightly smaller than that of X. The shapes are very similar. Some previous work has been reported on X-ray analysis of the hearts of twins. Verschuer and Zipperlen (53) found that a comparison of the roentgengrams of identical and fraternal twins demonstrated a significantly greater degree of similarity for identical twins, a finding which is supported by the study of Curtius and Korkhaus (12). In consideration of the errors in taking the teleoroentgengrams as well as in the actual measurement of the films, we conclude the differences in heart size are not significant, while the Lincoln-Spellman (27) norms indicate that the triplet hearts are approximately normal in size.

The sinus films are of particular interest for this study, being so nearly identical in shape as to make it difficult to distinguish between the three films. This finding is supported by Poole's (38) study of over 2,200 sinuses. He concludes that exactly similar sinuses are extremely rare. Some differences are noted in the condition of the triplet sinuses. The left maxillary of X shows definite evidence of infection, and a somewhat less marked infection of the right, both maxillaries of Z are infected, while both are clear in the case of Y.

The sella turcicas are practically identical, as is also the development of the mastoid process, though the left mastoid of Z is somewhat smaller than the other five. Very slight differences are present in the toes of the

triplets, the terminal phalanx of Z's little toe on both feet showing an epiphysis which is not present in the feet of Y and X. (Plate VII). Otherwise the epiphyseal development is the same in all six feet, as is the development in the knees, hands, elbows and shoulders.

There is a distinct difference in the ossification of the pelves. X shows a more marked union of the ischium and pubis on the left side than on the right, in the case of Z this finding is reversed, while Y shows a smaller degree of union on both sides. The pelvis of X appears to be entirely normal, but that of Y and of Z not entirely so. In the case of Y there is a definite occult spina bifida of the lamina of the fifth lumbar, and a fusion between a portion of the lamina and the beginnings of the spinous process of the fifth lumbar and the spinous process of the fourth. Z has a definite occult spina bifida of the first sacral, and a fairly marked lumbarization of the transverse processes of the first sacral, which are almost completely separated off. Z also shows a scoliosis and rotation of the lumbar spine which is not present in X and Y. Later comparisons of the backs of the triplets showed that all three also have a slight kyphosis of the lumbar spine. On the whole, the differences are less striking than are the similarities, and it is interesting to note that part of the time X and Y are most alike, at other times X and Z, or Y and Z. The results seem to indicate, that on the whole, Z has suffered a somewhat greater effect of any slight rickets, and after-effects of childhood diseases than have Y and X, which may, in part, account for her slightly inferior body build, particularly with respect to height.

Thirty six inch films were made of the wrists, care being taken to control the position of the hands. The eight ossification centers are all present in all six wrists, and in very nearly the same stage of development. (Plate VII) The writer has measured the area of the carpal bones by means of the so-called paper weight method, tracing the out-

TABLE 9

Ossification Areas of the Wrists of the Triplets

X	Right	5.77 Cms.
	Left	6.05
Y	Right	6.05
	Left	5.82
Z	Right	5.70
	Left	5.83

lines of the ossification centers on Eastman Kodaloid, cutting out the resulting outlines and weighing on an analytical balance. From these weights it was possible to calculate the area from the predetermined weight of the Kodaloid per unit area. (Table 9). Considering the errors of the determination one would conclude that the degree of ossification of the wrists is very nearly the same, although again, as far as the writer is aware, no norms of ossification areas of same-sexed siblings at comparable ages are available.

PHYSICAL EXAMINATION

The writer acknowledges the coöperation of Dr. Irvine McQuarrie who arranged to have the triplets hospitalized in the pediatrics department for two days. Under the direction of Dr. A. H. Fuller the staff made a rather complete physical examination of the triplets. The full report is too lengthy to reproduce and only a summary of the main items of interest is given. (Table 10). Unfortunately for the purposes of this study, X and Z were suffering from colds at the time of examina-

TABLE 10

Summary of General Physical Examination of Triplets

	X	Y	Z
Respiration	20	20	20
Pulse	118	85	85
Temperature	100.4	98.0	100.5
Blood pressure	95/60	90/65	95/60
Muscular system	Same degree of development		
Skin	0	Small mole left cheek	Naevus pig. on rt. scapula
Neuro-muscular	Slight choreiform movements		
Lymph nodes			
Ant. Cerv.	Palpable but soft		
Post. Cerv.	Not palpable		
Inguinal	Palpable but soft		
Head	Frontal bosses prominent		
Eyes and Reflexes	Normal	Normal	Normal
Ears	Imp. Ceru.	Ceru.	Imp. Ceru.
Nose	Normal	Normal	Normal
Tonsils	Slightly enlarged-noninjected		
Thorax	Asthenic—no rosary or grooves		
Hernia	0	Rt. Ing.	0

tion, and two days later X came down with an acute Otitis media. The results are therefore not strictly comparable. Despite this condition the findings for the triplets were quite similar. Rather extensive clinical comparisons of identical and fraternal twins have been made by Verschuer and Zipperlen (53) and Curtius and Korkhaus (12). A quotation from the work of the latter is of interest at this point. "Unsere Beobachtungen lehren, dasz trotz der Nachweisbaren morphologischen und physiologischen Identität eineiigen Zwillinge bei gleicher Exposition, verhältnismäszig häufig die konstitutionelle Disposition der beiden Partner gegenüber der infektiösen Noxe sich qualitativ und quantitativ stark unterscheiden kann." However, it must be noted that the usual clinical examination has for its purpose the discovery of pathology and that, therefore, the techniques used are not always suitable for investigating normal variation. The inguinal hernia of Y is a small one, and examination disclosed that both X and Z have weaknesses in the same region on both sides. The difference is not, therefore, of major importance.

LABORATORY TESTS

The results of the laboratory tests are presented in Table 11. Again it must be pointed out that the techniques used are not always reliable for the comparison of normal variations.

The outstanding differences noted are those for blood sugar and leukocyte count of Y, and, somewhat less marked, Y's higher metabolism reading. Granting the monozygosity of the triplets we have here a clear example of differences being caused by apparently quite similar environment. Of particular interest is the finding with respect to the blood grouping. The blood of the remaining siblings, parents and one grandmother was grouped by the writer. In every case the grouping was IV, a finding which was somewhat unfortunate for the purposes of this study. Orel (37) has reported a study of blood groups found in triplets and a similar study is being made by Dr. H. Hilleboe at the University of Minnesota. Blood group studies of twins have been reported by Wiechmann and Paal (56) and Schiff and Verschuer (44). The latter state: ". . . . unter den erbgleichen Zwillingspaaren kein einigen Fall vorkommt, in welchen die Partner eines Paares zu verschiedenen Blutgruppen gehören." It would therefore seem, that the comparison of blood grouping is an important check on the diagnosis of type of twins or triplets.

TABLE 11

Results of Laboratory Tests

	X	Y	Z
Urine			
Color	amber	straw	straw
Appearance	cloudy	clear	clear
Sp. Gr.	1030	1012	1010
Reaction	acid	acid	acid
Sugar	neg.	neg.	neg.
Microscopic	occasional blood cells	neg.	neg.
Blood			
Hemoglobin	80%	82%	81%
White cells	5300	6500	10,200
Polymorphonuclear	65%	48%	50%
Lymphocytes	34%	50%	48%
Eosinophiles	1%	2%	2%
Non-prot. nitrogen	27.2 mg. per 100 cc.	26.1 mg.	26.6 mg.
Sugar	70 mg.	78 mg.	92 mg.
Blood Group (Moss)	IV	IV	IV
Blood Wasserman (Kolmer & Kline)	neg.	neg.	no record
Basal metabolism	plus 8%	plus 6%	plus 12%

The electrocardiograph was also brought into use and the readings appear in Table 12. However, for the purposes of twin and triplet research, a study of the literature, particularly the paper by Lincoln and Nicholson (28), seems to indicate that electrocardiograms add but little to the diagnosis of type of twins or triplets, for the above mentioned authors state, "Resemblances were seen in seventy-five of the children in thirty-two families of our total group of forty families. The similarity was striking in many cases"; and again "There is often a marked similarity in the electrocardiograms of members of the same family."

EYE AND EAR EXAMINATIONS

Dr. C. Hymes of the University Hospital refracted the eyes of the triplets. The eyes were dilated with homoatropine and the retinoscope used to determine the correction necessary for 20/20 vision. It should be noted that for comparative purposes atropine should have been used for two or three days, and consequently the results cannot be considered

TABLE 12

*Electrocardiogram Readings of the Triplets**

	X	Y	Z
Rate	109	109	89
Sinus			
arhythmia	present	present	present
tachycardia	present	present	present
brachycardia	0	0	0
P-R interval	.12 sec.	.14 sec.	.13 sec.
R-T interval	.32 sec.	.31 sec.	.35 sec.
P_1	1.0 mm.	1.0 mm.	1.0 mm.
P_2	1.5 mm.	1.5 mm.	1.5 mm.
P_3	1.0 mm.	.5 mm.	1.5 mm.
QRS_1	.06 sec.	.05 sec.	.04 sec.
T_1	2.0 mm.	2.0 mm.	1.0 mm.
T_3	2.0 mm.	2.0 mm.	2.5 mm.
Rhythm	All three irregular		

* As read and interpreted by Dr. Lindquist of the University Hospital.

as accurate as the readings would tend to indicate. The error of a retinoscopic examination is probably in the neighborhood of .25 diopters, which would indicate that the refraction of the triplets is practically identical. Dr. Hymes also made certain measurements of the external features of the eye which are reported together with the refraction in Table 13.

Beckershaus (7), Schmidt (45), Wibaut (55) and possibly others have made ophthalmological comparisons of twins. They all agree that identical twins are more alike than are fraternal. However, Schmidt in examining seventeen pairs of identical twins found differences as high as 2.0 diopters, which seems to be quite a pronounced difference, partly accounted for, no doubt, by the inclusion of twins from the higher age levels.

The hearing of the triplets was compared by means of the Weston audiometer, the audiogram readings being recorded by Dr. Horace Newhart of the University Hospital (Table 14). Due to badly impacted cerumen in the right ear of Z, the reading for that ear was very low. However, one hour after removal of the impaction, another reading

was taken with results as shown. The error of measurement for the audiometer is unknown, as far as the writer is aware, but it is probably quite large, particularly when used on younger children. In this case

TABLE 13

Refraction and External Measurements of the Triplets' Eyes

		X	Y	Z
Corneal diameter	O.D.	11.2 mm.	11.2 mm.	11.25 mm.
	O.S.	11.2 mm.	11.2 mm.	11.25 mm.
Bimalar width		7.95 cms.	8.30 cms.	8.25 cms.
External canthial diameter		7.73 cms.	7.75 cms.	7.47 cms.
Internal canthial diameter		2.71 cms.	2.68 cms.	2.53 cms.
Palpebral fissure		2.54 cms.	2.50 cms.	2.57 cms.
Interpupillary distance		5.31 cms.	5.23 cms.	5.23 cms.
Projection of eye ball	O.D.	15.7 mm.	15.5 mm.	16.2 mm.
	O.S.	15.2 mm.	15.5 mm.	16.2 mm.
Esther (X)	O. D.			.37 Cyl. × 80
	O. S.	.25 Sph.		.25 Cyl. × 85
Ethel (Y)	O. D.	.12 Sph.		.50 Cyl. × 85
	O. S.	.25 Sph.		.37 Cyl. × 85
Esmer (Z)	O. D.			.25 Cyl. × 85
	O. S.			.25 Cyl. × 90

TABLE 14

Audiogram Readings of Triplets

		64*	128	256	512	1024	2048	4096	8192
X	Rt.	20**	20	20	20	10	5	5	20
	Lft.	15	15	15	15	5	5	10	35
Y	Rt.	15	15	15	15	10	10	15	20
	Lft.	10	10	15	15	10	10	10	30
Z	Rt.***	35	35	40	40	25	25	50	60
	Rt.	25	25	25	25	15	20	30	35
	Lft.	25	25	20	20	20	10	10	15

* Vibrations per sec.

** Degree of hearing loss expressed in sensation units.

*** The second reading for the right ear was taken one hour after removal of impacted cerumen.

the parallelism of the three individuals is marked. Macfarlan has reported an audiogram of one pair of identical twins with similar parallel results (29).

Dr. Newhart also examined the nasal passages of the triplets, but found no outstanding differences. The septa deviate slightly in all three, but the passages are clear. The nasal septa of twins have been made the object of an elaborate study by Schwarz (46). He found that identical twins are very similar when the nasal septa are compared, while for fraternal twins the differences may be very striking. In our case the sinus roentgengrams indicate a highly similar organization of the nasal septa.

TEETH

The teeth of the triplets were made the object of considerable study, and detailed findings will be published at a later date. Two sets of casts of the teeth have been made, the first by Dr. J. F. Knudson of Pelican Rapids, and the second by Dr. Jos. T. Cohen of the Institute of Child Welfare.[9] In addition, through the coöperation of Dean W. F. Lasby of the University Dental College, roentgengrams were made of the teeth. (Plate VIII).

The writer is indebted to Dr. Cohen for the following summary of the differences and similarities in the dental characteristics of the triplets.

X-Y

The dental arch of X is slightly wider than that of Y.
The teeth of Y are more decayed.
The eruption of the permanent teeth of Y is slightly advanced over that of X.

X-Z

The dental arch of X is wider than that of Z.
The teeth of X are less decayed.
Eruption of permanent teeth is about the same.

Y-Z

The dental arch of Y is larger than that of Z.
The progress of decay is about the same.
Eruption of permanent teeth is slightly more advanced for Y.

[9] Dr. Cohen is coöperating with Miss Howard, collecting dental casts of the teeth of the triplets.

Although differences are found, the similarity of the three sets of teeth is striking. A study of the roentgengrams, tooth by tooth, (Plate VIII) shows a very high degree of similarity, particularly when it is remembered that it is very difficult to secure exactly the same angle for the three roentgengrams of each tooth. Examination of the dental casts corroborates the finding of a high degree of similarity (Plate IX) both with respect to occlusion and the general physical appearance of the teeth and arches. The findings are in general accord with those of two rather extensive studies of the dental characteristics of identical twins (Bachrach and Young, 5; and Goldberg, 21). A study of Goldberg's paper indicates that dental comparisons are quite reliable in the study of twins, and presumably also in the case of triplets. He states, "Except in individual cases where abnormal habits have interfered with intrinsic forces, the arch form, previsioned in the germ cells, is carried to fruition in the adult, and there is very little justification to assume that environment, either intra-oral or extra-oral, under average conditions in life, plays a very great rôle."

PSYCHOLOGICAL TESTS

The Stanford Revision of the Binet Simon Intelligence test was administered by the writer early in the spring, and in October by F. H. Finch, Psychologist at the University High School. The Kuhlman intelligence test, as well as other psychological tests, was administered by Miss Howard of the Institute of Child Welfare[10] (Tables 15 and 16).

TABLE 15

Results of Intelligence Tests Administered to the Triplets (I. Q.)

TEST	ADMINISTERED BY	X	Y	Z
S-Binet	Lund	109	104	104
S-Binet	Finch	106	108	107
Kuhlman	Howard	112	109	116
Goodenough Drawing	Howard	130	70	121

[10] Miss Howard is completing a psychological study on approximately twenty sets of triplets.

TABLE 16

Results of Various Psychological Tests Administered by Miss Howard

TEST	X		Y		Z	
Anderson-Page Tracing Test—	Time	Errors	Time	Errors	Time	Errors
A	78	5	21	26	31	10
B	51	5	18	29	32	10
C	46	8	15	24	29	7
D	31	3	22	14	27	3
Card Sorting Test—						
Av. time for 15 trials	54 sec.		48 sec.		41 sec.	
Spool Packing Test—						
No. trays filled in 3 minutes:						
Trial 1	82		108		108	
Trial 2	88		106		106	
Arthur Performance Tests—						
Knox Cube	8.5		10.		8.	
Seguin Test	16.		16.		21.	
Maze Test	18.		8.		15.5	
Kohs Block Test	43.		19.		49.	

The only striking difference in the I. Q.'s as measured by the tests used is that of the Goodenough test result for Y. However, a possible explanation is that Y took the test as more or less a joke and did not attempt to do her best. Outside of that discrepancy, the results are very similar, and probably the differences can be considered as smaller than the error of the tests employed. Interestingly enough, the responses of the triplets were quite different to the separate parts of each test, and still the results on the tests when totalled show great similarity.

The results obtained on the psychological tests (Table 16) are difficult to interpret. It seems fairly obvious that in their psychological reactions the triplets may, on occasion, be quite different. This is particularly striking for the Spool Packing test, where the results for X are very much lower than those for Y and Z. Again on the maze test, Y is lower than X and Z. Allowing for such variations we find that the results are on the average very concordant, and probably well within the limit found for identical twins.

The writer, with the help of Alice Johnson, administered a number of standardized educational tests. (Table 17).

TABLE 17

Results of Various Educational Tests *

TEST		X	Y	Z
Van Wagenen Spelling—				
Scale A—Division	1	39.0	40.5	48.0
	2	49.5	37.5	50.5
Scale B—Division	1	40.0	40.0	41.0
	2	40.5	40.5	48.0
Scale C—Division	1	42.0	42.0	47.0
	2	43.0	41.0	37.0
Scale D—Division	1	44.5	40.0	45.5
	2	46.0	44.5	51.5
Scale E—Division	1	44.5	41.5	45.5
	2	46.5	40.0	47.5
Chapman-Cook Speed or Reading Test—				
Form A		20.0	17.0	21.0
Form B		22.0	19.0	18.0
Thorndike-McCall Reading—				
Form 1		59.0	59.0	61.0
Los Angeles Reading—				
Form 1		24.0	21.0	28.0
Gates Silent Reading—				
Gen. Significance—Form 2		87.0	78.0	81.0
Directions—Form 1		68.0	87.0	63.0
Woody-Van Wagenen Arithmetic—				
Scale S1		37.0	43.0	46.0

* All results expressed in score points.

The results obtained with the educational scales used are again difficult to interpret. On the whole, the resemblances are more striking than are the differences. X's score on the arithmetic scales is considerably lower than that of either Y or Z. Again on one form of the Gates Silent Reading test we find X and Z very similar, while the score of Y is very much higher. The scores obtained on the Van Wagenen spelling scales are particularly striking in their similarity, the range of each girl on her series of tests being: X-10.5 score points, Y-7, and Z-10.5, while the range from the lowest triplet score to the highest (X lowest to Z highest) is 11.5 score points. In other words, the total range of scores for each individual is practically the same as the total range for the three individuals. The general results obtained from the tests used are

supported by the evidence of the report cards of three different teachers, for grades 1, 2, and 3. An analysis of the report cards indicates that the triplets are very similar in their school work, although some difference is apparent; for, even in the marks on deportment and industry differences are apparent, in one or two cases being as much as one letter grade. It is granted, however, that teachers' marks cannot be considered reliable enough to make such a finding very important. It is, on the other hand, additional evidence to indicate that seemingly identical individuals, insofar as heredity is concerned, may react quite differently to a common environment.

METHODS OF DIAGNOSING IDENTICAL TWINS

The Siemens (47) dermatological method of diagnosing one- and two-egg twins, was applied to the triplets by Dr. E. Boyd, Dr. H. Hilleboe, and the writer. The method has been carefully worked out and Dr. Siemens states, "An actual *mistake* [11] in determining the identity of twins need never occur (47)." The use of this method may, however, be unreliable in the hands of those inexperienced in its use. In the case of the triplets, the result of this comparison was favorable to the conclusion that the triplets are of the one-egg type, the only discordant finding being that of the presence of a naevus pigmentation on the right scapula of Z, and a small mole on the right cheek of Y, neither of which were found on the other two individuals. The ear comparisons deserve special mention. In general appearance, size, and conformation, the ears are practically identical. However, both auricles of Y stick out from the head, while for X only one sticks out, and for Z both lie close to the head as does the left of X—a most interesting condition of asymmetry.

Stocks (51) has worked out two methods for diagnosing the type of twins, and his first method was applied to the triplets by the writer. The method consists of a comparison of the finger prints of the left hands with the lefts and the rights with the rights. If on seven or more of the homolateral digits the patterns are substantially identical the twins are to be considered identical. In case of doubt, further comparisons are made on the measurements of height, head length, breadth, and circumference and the interpupillary distance, three of which differences must not exceed certain limits given by Stocks. Table 18 gives the findings in the case of the triplets.

[11] Italics are mine.

TABLE 18

Results Obtained When the Stocks' Method of Diagnosing Twins was Applied to the Triplets

CRITERION	XY	XZ	YZ	XYZ
Number of homolateral fingers with patterns practically identical	8	7	7	5
Is difference in height less than 6.3 cms.?	Yes	Yes	Yes	Yes
Is difference in head circumference less than 1.3 cms.?	Yes	Yes	Yes	Yes
Is difference in head length less than .55 cms.?	Yes	Yes	Yes	Yes
Is difference in head width less than 1.3 cms.?	*No*	Yes	Yes	*No*
Is interpupillary difference less than .3 cms.?	Yes	Yes	Yes	Yes

As long as only one discordant finding is reported the triplets fulfill the Stocks criterion, although it is granted that this method has been devised for use on twins only, and its application to triplet data may be questionable.

Stocks' alternate method was also applied, and here again the results are in accord with the conclusion that the triplets fulfill the requirements for being classed as three pairs of identical twins on the basis of the X-Y, X-Z, and Y-Z comparisons.

SUMMARY

The main purpose of this study has been to establish whether or not this set of triplets can be regarded as having been derived from one egg and therefore being monozygotic. Three criticisms of the study are apparent. Positive placental evidence is lacking which, in the opinion of the writer makes it impossible to draw a final conclusion. Again, the comparisons which have been used have been originated and standardized largely on twin material, and at the present time our knowledge of the interrelationships of the various variables of triplets as compared with those of twins is very meager, necessitating caution in interpreting the findings. Finally, for most of the comparisons made, the variation normally to be expected in ordinary siblings of the same sex at comparable ages is unknown, making the drawing of conclusions a hazardous

undertaking. In spite of these shortcomings, the comparisons made have been so numerous, and so heterogeneous, while the results have been so consistent that the probability that the triplets are really monozygotic is very high. A similar detailed study of another set of triplets, where a number of siblings are available for comparison, and where positive placental evidence can be obtained, should, it would seem, be all that is necessary to establish the possible one-egg origin of triplets.

The various comparisons which have been used have been divided into three categories by the writer, and may be summarized as follows:

Evidence considered directly in favor of monozygotic origin:

1. Hair and eye color
2. X-ray films of sinuses, mastoids, and epiphyseal development
3. Bodily dimensions
4. General body build and appearance
5. Hands
6. Ears
7. Finger, palm and sole prints
8. Eye measurements
9. Dental characteristics
10. Stocks' criteria

Evidence which is less direct and certain, but, nevertheless in favor of monozygotic origin:

1. Single placenta (?)
2. Weight record
3. Blood grouping
4. Physical and mental developmental history
5. Results of mental tests
6. Nasal septa
7. Hair whorls
8. Educational tests
9. Refraction
10. Audiograms
11. Siemens' comparisons

Evidence, which though not negating monozygosity, is difficult to interpret:

1. General physical examination
2. Laboratory findings
3. Electrocardiograms
4. Psychological tests

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PLATES

LEGENDS FOR PLATES

PLATE I. The J. N. Johnson Family. Reading from left to right. Top Row: Anna, Alice, Albert, Edna, and Lillian. Bottom row: John, Father, Esmer (Z), Ethel (Y), Esther (X), Mother.

PLATE II. Esmer, Ethel, and Esther. 9 Months Old (Above) and 3 Years Old (Below).

PLATE III. (Above) Esmer, Ethel, Esther—7 Years Old; (Below) Present Profiles of Triplets—Esther, Ethel, Esmer.

PLATE IV. Finger Prints of Triplets. X—right, left. Y—right, left. Z—right, left.

PLATE V. Palm Prints of Triplets. Rights—Esther, Ethel, Esmer. Lefts—Esther, Ethel, Esmer.

PLATE VI. Sole Prints of Triplets. Esther—Right-Left. Ethel—Right-Left. Esmer—Right-Left.

PLATE VII. Roentgengrams of Triplets' Toes (Above) and Wrists (Below). Right—Esther, Ethel, Esmer. Left—Esther, Ethel, Esmer.

PLATE VIII. Roentgengrams of Triplets' Teeth. Uppers—Esther, Ethel, Esmer. Lowers—Esther, Ethel, Esmer.

PLATE IX. (Above) Dental Casts of Triplets Showing Occlusion; (Below) Dental Arches of Triplets, Esther, Ethel, Esmer.

PLATE I.

PLATE II.

PLATE III.

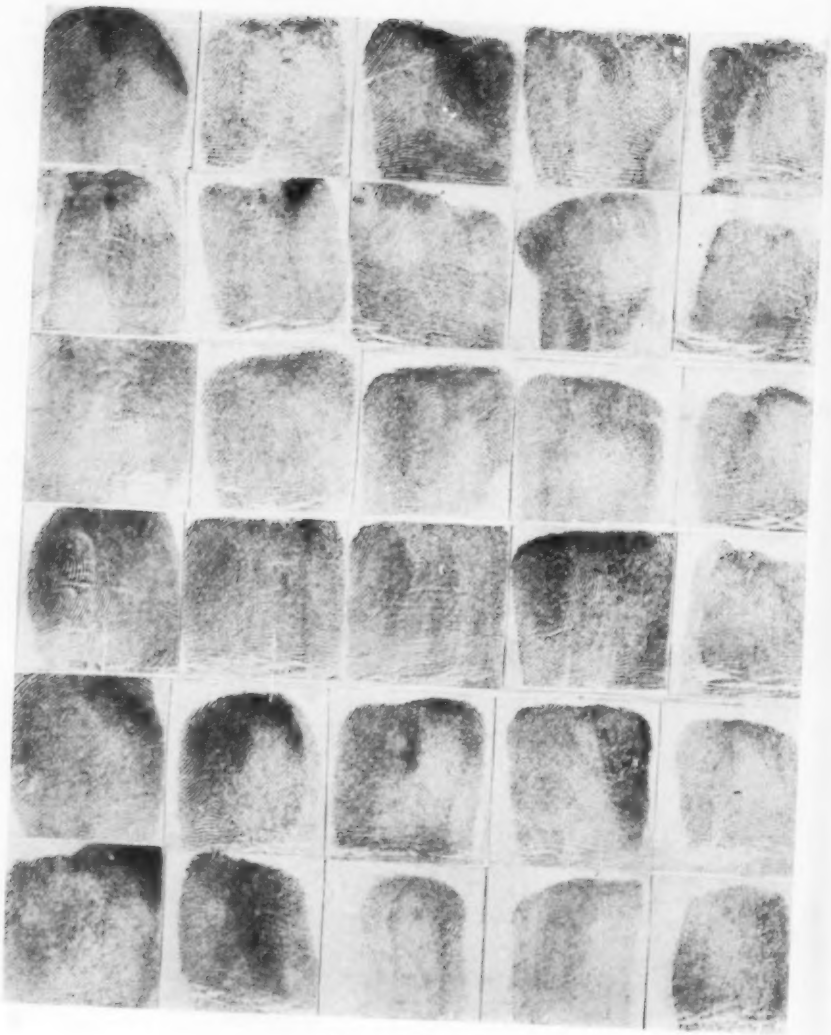

PLATE IV.

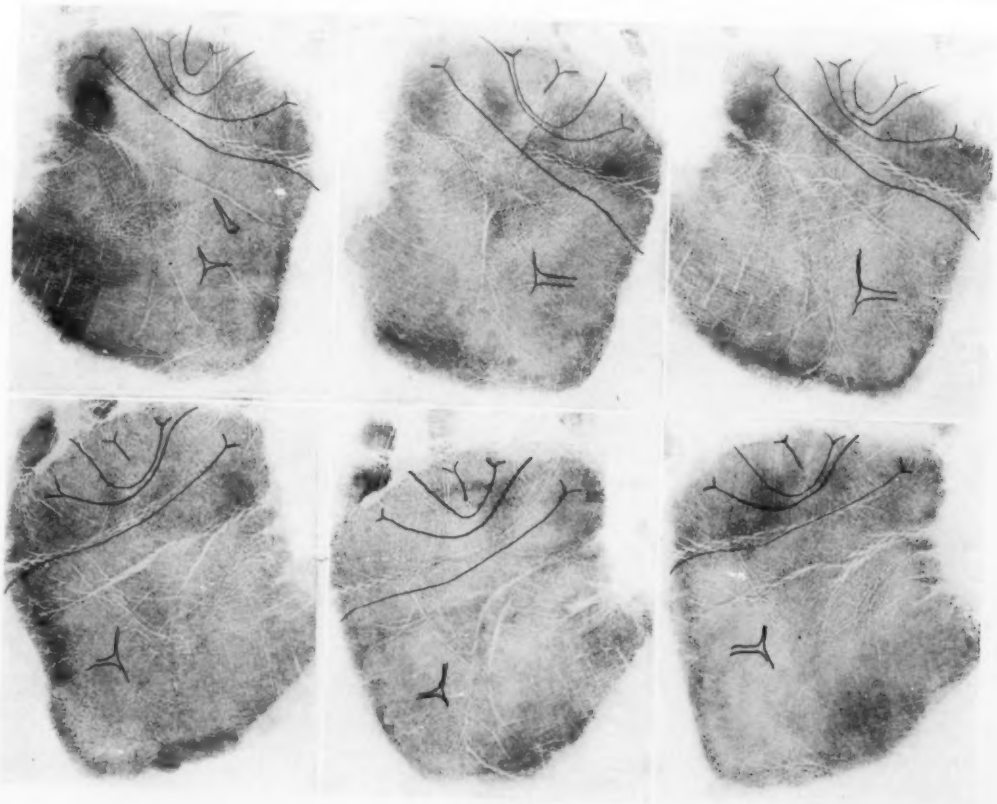

PLATE V.

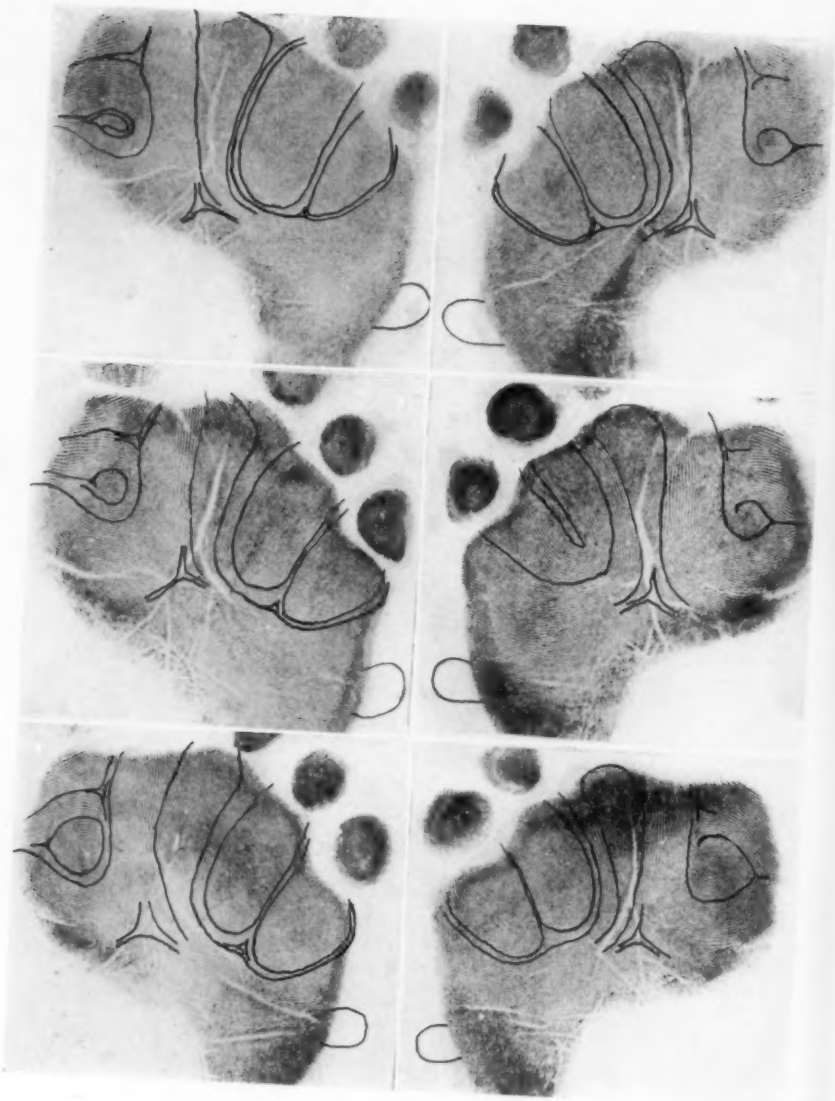

PLATE VI.

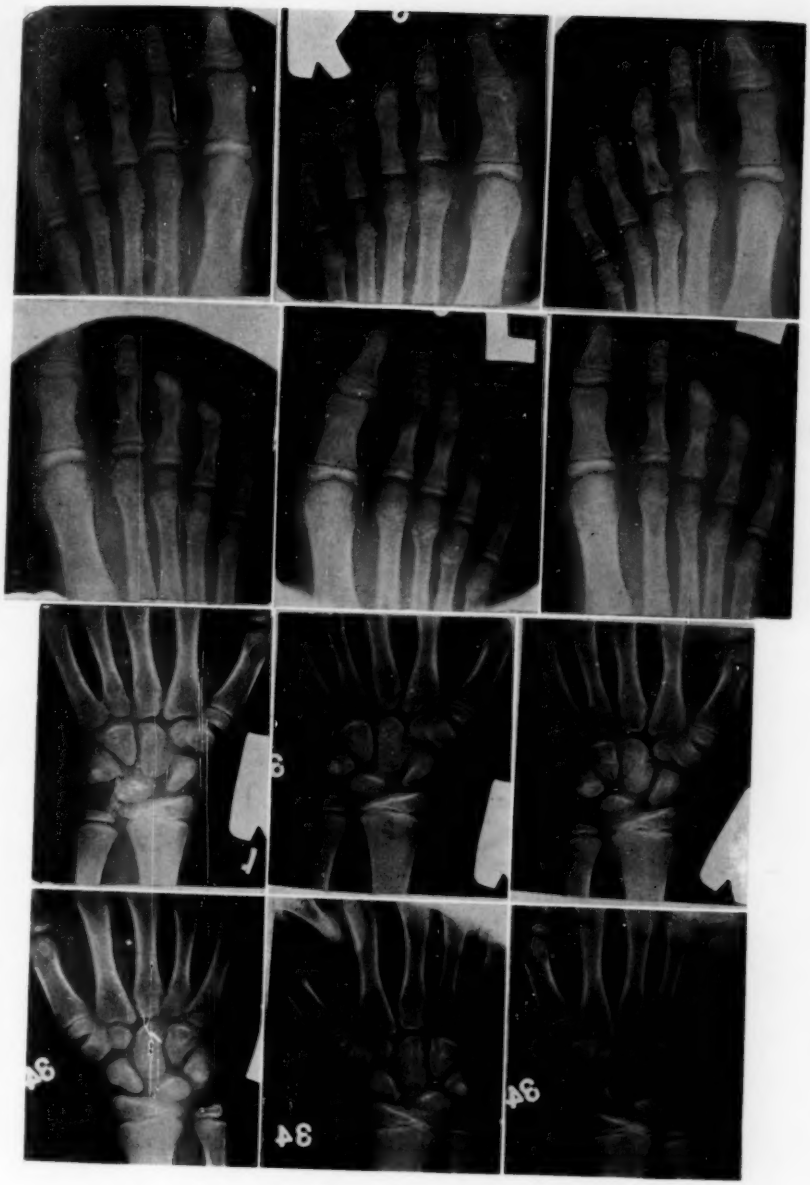

PLATE VII.

PLATE VIII.

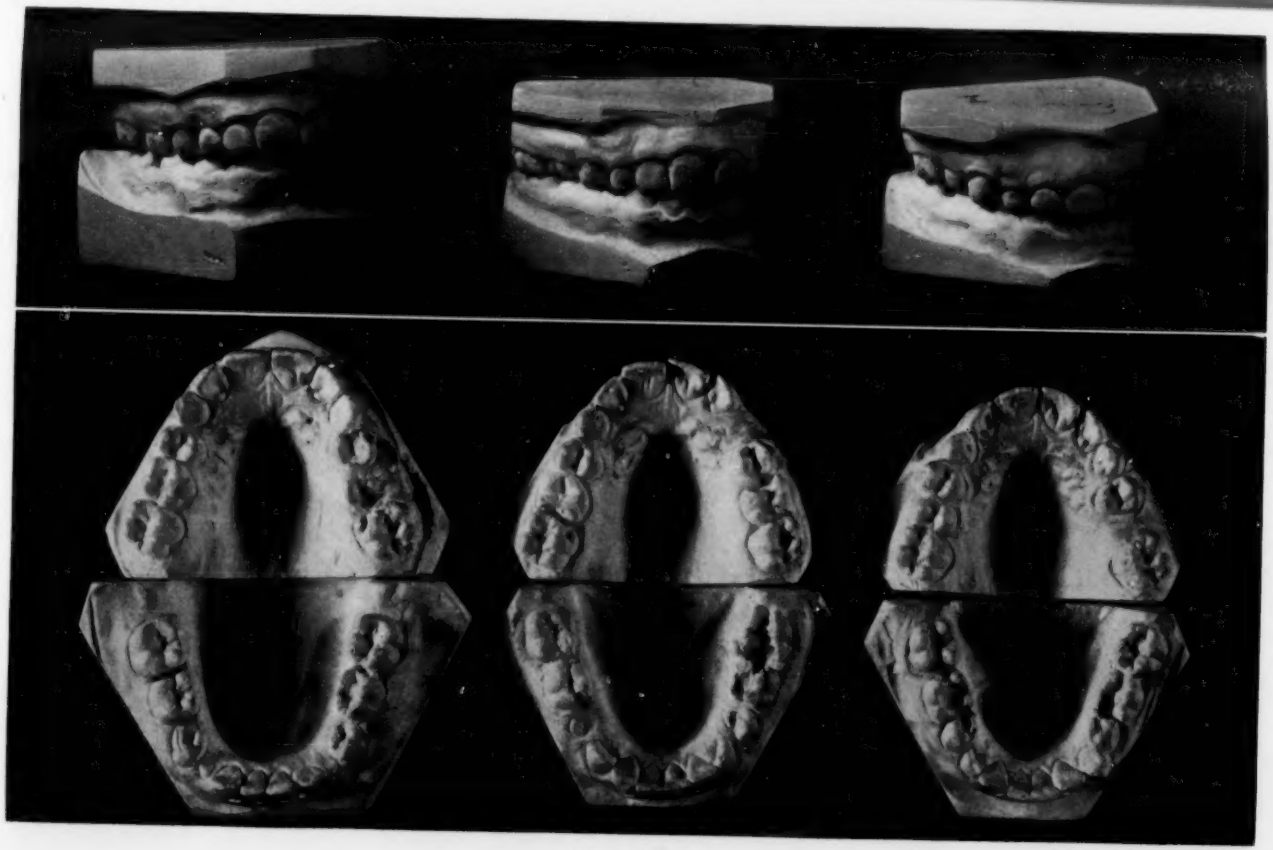

Plate IX.

THE FECUNDITY AND FERTILITY OF EARLY MAN

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To the student of the broader aspects of human population growth no fact stands out with greater distinctness than the rapidity and volume of that growth in recent times. Throughout historical time, to be sure, populations have increased and waned, as in Mesopotamia, Greece, Rome, Gaul, and the Iberian peninsula, as they did in half-veiled periods of prehistory and forgotten civilizations like those of Central America, Yucatan, and Cambodia. Doubtless demographic movements, here increase, there decrease, were taking place during the hundreds of thousands of years of man's early career. It is possible that locally and for relatively brief periods there may have been rates of increase comparable to those which, since the beginning of the nineteenth century, we have fallen into the habit of taking as a matter of course; but such rates could not have been long sustained, for the simple reason that in the long lower hunting stage of culture man's technical command over the resources of nature was not sufficient to support more than an exceedingly low density of population. It is safe to say that never before have the mass and volume of increase—the increase in absolute numbers—been remotely comparable to their magnitude in the past two hundred years, and especially in the past century; it is highly probable, also, that modern rates of increase are unique.

The population of Europe increased from about 185 millions in 1800 to 266 millions in 1850, and in the next seventy-five years to over 500 millions. In the meantime Europe sent out over 50 million emigrants, to accelerate the growth of "colonial" countries like the United States, Canada, South Africa, South America, and Australia. Such a phenomenal autogenous growth and expansion of European population was made possible not only by the progress of modern medicine, hygiene, and public health service, but by the availability of vast areas of fertile land for settlement, the advent of cheap land- and ocean-transportation, and the development of modern manufacturing technique and organization. Capitalism, in its technological and organizational aspects, the exploita-

tion of natural resources with all the arts of modern applied science, and modern population increase, are thus closely intercorrelated. Phenomenal increase in population has taken place in other than capitalistic countries also. The probable population of China at the end of the seventeenth century was but little over 100 millions. By the end of the eighteenth century it had increased to between 250 and 300 millions. The period of the massive increase of the Chinese population was thus about a century earlier than that of Europe.[1] The population of the Dutch Possessions in the East Indies (Java and Madura) increased from 16 millions in 1850 to 32 millions in 1925. The population of Argentina more than doubled between 1900 and 1925—from 4.7 millions to 9.9 millions. Asiatic Russia is estimated to have increased from 6 millions in 1850 to 22 millions in 1900 and 29 millions in 1925. The world population is estimated to have increased from 775 millions in 1800 to 1,564 millions in 1900 and about 1,900 millions at the present time.[2]

THE STATIONARY POPULATION OF EARLY MAN

The weighted average of the rates of increase, 1906 to 1911, for twenty-six countries for which Knibbs could find reliable statistics, was 11.59 per thousand, annually. This would double the population in sixty years. Knibbs estimated that at the probable rate of world increase, 1909 to 1923, world population would double in 105 years. It is obvious not only that such a rate of increase cannot continue long into the future, but also that it cannot have existed long in the past. Starting from a single pair, mankind would have to double only thirty times to reach a population of 2,147,800,000, a number materially in excess of the present world figure. At an increase of 1 per cent a year, this figure would be reached in a little over two thousand years. On the assumption that man has been on the earth a million years, his rate of increase during that time at large has been only 1-500th of 1 per cent a year.[3] If we broaden the term man to include not only *Homo sapiens* but *Pithecanthropus, Eoanthropus,* and *Sinanthropus,* and accept the present tendency to push man's origin far back to the Pliocene, it is obvious that

[1] Cf. Sir G. H. Knibbs, The Shadow of the World's Future, 1928, p. 53.

[2] Alois Fischer, Zur Frage der Tragfähigkeit des Lebensraumes, I Teil: Die Erdbevölkerung in ihrer bisherigen Entwicklung, *Zeitschrift für Geopolitik,* 1925 (II), pp. 766-769. The figures, in the absence of censuses, especially for the earlier years, are estimates.

[3] Knibbs, *op. cit.,* p. 11; The Mathematical Theory of Population, 1917, p. 31.

the average rate of increase must have been very much less than this. In any case, it is clear that throughout man's career, down to neolithic times, which on a proper time-scale are exceedingly recent, the human population as a whole was practically stationary.

The entire human population of the earth was very small and its average density very low. In the hunting stage men could not be otherwise than few and far between. The number of people that can find subsistence on a given area, in a given type of culture, is definitely limited, and the lower the stage of culture the more rigorous this limitation is. Throughout all the hundreds of thousands of years of prehistory, most of it before palaeolithic time, when human culture was advancing at imperceptible pace, the population remained very thinly distributed. Doubtless from the beginning man sought out, by a kind of blind tropism, the most favorable places. In an embryonic stage of culture he was able to survive only in such places. As the ages rolled by, and human culture gained a little advance, man, impelled by increase of numbers in the favorable regions, by climatic changes, and perhaps by the marauding propensities of peoples a trifle more advanced and in possession of superior weapons, pushed out wave after wave of migration, penetrated into more inhospitable regions, and succeeded in surviving in them. Occupancy of the least favorable habitats must have been comparatively recent. These regions could not be permanently occupied by peoples not in possession of a comparatively high technique of coping with their natural environment. This applies preeminently to the Eskimos. The higher and more barren mountain regions, the malarial morasses, the fever-ridden *tierras calientes,* the dense tropical jungles and rain-forests, and the deserts, have never, in all probability, had more than exceedingly sparse populations.

However far back we go in the demographic history of man, therefore, we should expect to find the human population unequally distributed, clustering here and there in nebulous nodules or streaming out along the seashore and migration "corridors" or resting thousands of years at a stretch in the river valleys where game and possibly wild cereals were abundant. Such is the evidence, for example, at Solutré, in the Rhone valley, "where the horse seems to have been the favorite food," and where "the broken bones of these animals, left as the refuse of many feasts, form a mass of breccia considerably over 100 yards in length

and in places as much as ten feet thick."[4] Under such conditions there may have been, locally, population densities considerably higher than that which on the average we can assign to hunting peoples.

THE DENSITY OF HUNTING POPULATIONS

Only rough approximation to the probable and permissible densities in the hunting economy can be made. The total number of a given people can usually be determined with no great accuracy, and it is difficult to ascertain with certainty the limits of the territory from which they obtain their subsistence. Ratzel, who gave considerable attention to the correlation between type of culture and density, held that density among hunting and fishing peoples is much too great if it is as much as one person per square kilometer, or roughly 2.5 per square mile.[5] The Tasmanians, who had no knowledge of metals and possessed only very rude stone implements, probably did not exceed 2000 to 3000 in number when they were discovered by white men. On an area of about 26,000 square miles the probable density was one person to from 9 to 13 square miles.[6] The Patagonian Indians, according to Ratzel, had over 200 square miles per capita, the Australians 42 square miles, and the Ojibways, who supplemented their diet with wild rice, 6 square miles. Generalizing, Ratzel arrives at the following scale:

> Hunting and fishing peoples in the Arctic: 75 to 200 square miles per capita.
>
> Hunting peoples in steppe and semi-desert regions (Bushmen, Patagonians, Australians): 45 to 200 square miles per capita.
>
> Hunting peoples with some agriculture or trade with agricultural tribes (Dyaks, Papuans, the poorer negro tribes of central Africa): ½ to 2 square miles per capita.

To fisher folk in North America and Polynesia he allows densities up to 4.5 per square mile, and to shepherd nomads 1.8 to 4.7 per square mile.[7]

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[4] W. J. Sollas, Ancient Hunters, 3d ed., 1924, p. 365. Cf. Sir Arthur Keith, New Discoveries Relating to the Antiquity of Man, 1931, p. 321.

[5] Anthropogeographie, 4th ed., Vol. II, 1922, p. 167.

[6] M. L. Newbegin, Man and His Conquest of Nature, 1912, p. 48.

[7] *Op. cit.*, pp. 168, 173. The figures given above are approximate conversions of Ratzel's figures per square kilometer. S. H. Roberts (Population Problems of the Pacific, 1927, p. 86) estimates the number of native Australians at the time of their discovery by whites at 160,000. This would give 18 square miles per person—a much higher density than Ratzel's estimate.

Wissler, on the authority of an unnamed "special student of the subject," states that from 300 to 400 square miles are required to feed an Eskimo family.[8]

Utilizing Mooney's painstaking estimates of the Indian population north of Mexico at the time of exploration by white men, Wissler gives the following estimates, among others, of the aboriginal Indian population in the United States and Canada: California, 0.6 square mile per person; southern plains, 9 square miles; central mountain region of the west, 22.4 square miles; central and eastern Canada, 24.1 square miles. For the great central plains region, estimated at 835,000 square miles, he allows 7 square miles per capita.[9] These densities agree fairly well with Ratzel's estimates, though comparison is difficult. The extremely low density of the aboriginal Indian population is evident. Only in California did it approach 2 per square mile. Kroeber allows, even for California, only 1 per square mile.[10]

We may assume that in past ages, before man with weapons had had time to disturb the primitive balance of nature, game was more abundant than among the extant peoples to whom the above estimates apply.[11] All the modern "savages" with whom the white man has come in contact have, with few exceptions, been better equipped with weapons, probably, than most even of the best of palaeolithic hunters. Technological advance and diminished game supply may be taken as counterbalancing each other. We may suppose that for an indefinite time back into the past, a rough equilibrium was maintained between the sources of subsistence, on the one hand, and man's cultural equipment and his net fertility, on the other. This broad generalization may not be strictly true to the facts at all times and places, but it is a necessary postulate for an understanding of the ages of practically changeless culture and stationary population.

THE FECUNDITY OF PRIMITIVE PEOPLES

Once those regions habitable in a given state of culture are populated up to the limit possible in that stage, it is obvious that further increase

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[8] Man and Culture, 1923, p. 343.

[9] An Introduction to Social Anthropology, 1929, p. 31. Cf. James Mooney, The aboriginal population of America north of Mexico, *Smithsonian Miscellaneous Collections*, Vol. 30, No. 7, 1928.

[10] Handbook of the Indians of California, *Bureau of American Ethnology, Bulletin* 78, 1925, p. 490.

[11] Cf. Karl Kautsky, Vermehrung und Entwicklung, 3d ed., 1921, p. 150.

can take place only through an advance in culture, which will either permit migration to less favorable regions or enable more subsistence to be obtained in the old one. It is a fundamental generalization in the theory of population that the size of the human population and the rate and momentum of its growth or decline are closely dependent on the state of culture and the rapidity of its evolution. Not a few students of population, however, think that there are even more fundamental determinants, of a biological nature, influencing the growth or decline of human populations.

It is thus an interesting question whether primitive population was kept in equilibrium with natural resources by cultural influences or by some biological factor. During all the ages of stationary population, was the human species kept in balance with its subsistence resources by the practically stationary character of its culture, its technique of food-getting, and by the tremendous and multiform lethal factors to which primitive man was subject, or were there fundamental physiological or biological influences in the nature of the early human organism itself which contributed significantly to that balance? The correlation between culture and population is so obvious that it would be unnecessary to raise this question, had not Carr-Saunders in his book, *The Population Problem*, 1922, widely cited by both anthropologists and sociologists, advanced the theory that the fecundity of primitive peoples was lower than that of modern populations.

It is necessary at this point to call attention to the technical but essential distinction between fecundity and fertility. Fecundity is the power of procreation. Fertility is the degree to which that ability is realized. In woman, fecundity is the physiological capacity to bear living children, and fertility is measured by the number of living offspring actually borne. High fecundity will result in high fertility, if the capacity to conceive and give birth to living children is not interfered with in one way or another by cultural influences. Disinclination to have offspring because of rational considerations, whim, or institutional pressures, does not affect fecundity. The fecundity of a woman who bears few or no children may be just as high as that of a woman who bears many.

It is desirable, also, to distinguish between a natural, inherent, and perhaps inherited low fecundity, and a low fecundity or infecundity induced or acquired by reason of environmental factors. A woman, or a man, may be fated to sterility from birth; or sterility may be the result

of accident, disease, or physiological exhaustion and maladjustment. Sterility thus caused is obviously the result of environmental factors; it is not inherent in the organism *per se*. A large amount of sterility, for example, is due to acquired venereal disease. It is possible that a given family strain may have a low fecundity, but there is no scientific evidence that differences of natural fecundity exist between the various races of man.

Distinction should also be made between gross fertility and net fertility. The fertility-rate is usually defined as the number of live-births, annually, per thousand women of child-bearing age. It may also be measured by the number of live children born to each woman, or to each thousand women in the course of their reproductive period. Gross fertility is the number of live-births per thousand women all of whom live through the reproductive period, that is, from the age of 15 to that of 45 or 50. Some girls die before the age of puberty, and some women before completion of the reproductive period of life. Making allowance, by use of a mortality table, for these premature deaths, we get the net fertility—the number of offspring borne by those of 1000 girl babies who survive to have children of their own.[12] This is a correct method of calculating net fertility when we wish to know how many children the women of a given generation contribute to the next generation, and to predict the trend of autogenous growth or decline of population.

It is axiomatic that if a species is to survive, the number of births, generation after generation, must be at least equal to the number of deaths. Otherwise succeeding generations will be smaller, and in the course of time the species will die out. The fact that the human species has survived, over hundreds of thousands, if not millions, of years, shows that at least an average equilibrium of births and deaths was maintained. Furthermore, the fact that mankind, starting from very small numbers, if not from some primitive Adam and Eve, expanded over the habitable earth, to become the most widely distributed of all species, proves that the fertility of early man was high enough to provide from time to time an excess of births over deaths, and thereby an increase in numbers sufficient to make this expansion possible. The primitive migrations by which in the course of time man spread to the remote four corners of the earth, and occupied all the habitable lands between, could not have taken place on any other terms. Once the expansion of

[12] Cf. R. R. Kuczynski, The Balance of Births and Deaths, Vol. I, North and West Europe, 1928.

primitive hunters over the earth had been accomplished and the land filled to capacity, that is, to the low density which it would tolerate under a primitive hunting economy, population would settle down, broadly speaking, to stationary numbers, in equilibrium with the natural resources at its disposal and utilizable by its primitive technique of getting a living.

POPULATION AND THE FOOD SUPPLY

The balance between early man and the food supply constituted an extreme case of "natural economy," and one which lasted incomparably longer than any other type of economy man has ever lived under. We may suppose that even in the absence of any but the most rudimentary implements and weapons, the first groups of the human species, aided no doubt by favorable geographic environment, a climate not too rigorous, and a fairly adequate food supply, pushed out into, and occupied, contiguous territory. Such natural overflowing would last as long as conditions were favorable to increase of numbers, and as long as there were near-by favorable regions unoccupied. What may be called contiguous expansion would continue, without special stimulus from climatic or other geographical changes. When, under such circumstances, a natural balance was struck between population and natural resources in a given region, the positive check of famine or starvation would not act vigorously, because the excess population could and would drift out into regions in which game and other food resources, being hitherto unexploited by man, were still plentiful.

When all these available contiguous territories were occupied, however, further increase of population would bring the pinch of hunger, and war—conflict for territory and subsistence—would be a natural result. If climatic changes made an inhabited region markedly less favorable, great waves of exodus would be instituted, and those great pressures of tribe on tribe, race on race, which are supposed to lie back of the distribution of racial types over the earth, would have been started. During these great climatic changes and migrations, there could be no permanent balance of nature established, although the changes may have been so gradual that their effects could be observed only at intervals of centuries or of thousands of years. For shorter periods, in which climatic changes and resultant shifts of fauna and flora were too slight to be perceptible, there might be an apparent equilibrium between population and subsistence resources.

In the earlier stages of man's career, it was probably not so much food supply as it was man's relative defenselessness against other animals and the forces of nature which limited his numbers and retarded his increase. We may guess that for hundreds of thousands of years prior to and during the lower palaeolithic stage, man was almost as much hunted as hunter. To the extent that this was true, his fertility had to be high enough to make good the losses from predatory carnivores and other lethal environmental factors. During this long phase of the struggle for existence, man entered the lists with only one advantage, and that at the beginning comparatively slight—his incipient reasoning capacity. The balance of life and death turned not only on his prowess and skill in finding and killing game, but on his circumspection and cunning in avoiding being eaten himself—and on a fertility high enough to counterbalance the dangers he could not escape.

Theoretically, the best possible balance in a hunting economy would be had when no game animal dies a natural death—when all that die, unless killed by beasts of prey, are killed by man for food and clothing. Such conditions would set the maximum population density possible to a pure hunting economy. Man can indeed raise this maximum by killing off predatory animals, as the western stockmen do to-day, but if the human population thereupon increases, a new balance is merely set up, without any improvement in the standard of living. Primitive man cannot eat his cake and have it too, any more than modern man can. The game supply is his capital, and if he does not sense the wisdom of conserving it he suffers, like any other thoughtless spendthrift.

It is probable that in the course of time practically all hunting peoples increased in numbers until further increase was more or less summarily stopped by dwindling game supply. "Everywhere," says Wissler, "the Eskimo live up to the food supply. The Indian hunters of eastern Canada and the corresponding parts of the United States were also in a state of equilibrium. . . . . In general, it appears that all hunting cultures, long sustained, show a balance between fauna and population. If too many game animals are killed one year, a proportionate number of persons starve during the next." [13] This suggests the existence, in the hunting stage, of more or less regular rhythms, ebbs and flows, of population—rhythms which would not entirely disappear until the domestication of milk and meat animals made grazing possible, or until the beginnings of primitive agriculture, in neolithic times, afforded a more

[13] Man and Culture, 1923, p. 343.

reliable food supply. In the hunting stage man enters the economy of nature in much the same way as other animals. Thompson-Seton, on returning from a journey down the Mackenzie river some years ago, described the ebb and flow of the rabbit and fox populations of northwest Canada. When the rabbits increase the foxes wax fat and multiply, until they have eaten most of the rabbits. Then the fox population dies off of starvation and gives the rabbits a chance to multiply once more, and the cycle begins again. This type of oscillation is well known to naturalists. If cycles of this kind occurred in the human species, they were short period oscillations, producing little or no effect on the long-run movement of the human population. This remained, after the primitive migrations had spread man over the earth, practically stationary and of very low density. The stationary population and low density can be adequately explained by the limited food supply, the low state of culture, and the inevitably high mortality rates.

THE DEATH RATE OF EARLY MAN

Theoretically, of course, a stationary population may be due either to an excessively high mortality rate or to a low fertility rate. Conceivably, low fertility might be found under conditions which would compel us to attribute it to low fecundity, but there is nothing to force us to such conclusion in the case of early man. In fact, there is nothing which clearly indicates that early man did not have both a high fecundity and a high gross fertility. It is certain, however, that a high ***net*** fertility did not exist. A net fertility high enough to provide some increase of population could be looked for only in those rare times in which cultural mutations of some magnitude were in progress—advances which would either diminish the dangers to which human life was exposed or render possible a significant increase in the food supply. Such a cultural mutation, for example, occurred in our own time with the discovery of the cause of germ diseases, serum treatment, and the general advance of medical knowledge.

A cultural mutation to a higher technique of capturing and killing game would enable the early hunter to secure an ampler food supply and increase his numbers, so long as he did not, by killing too many animals, reduce the net fertility of the game population to a point too low to maintain that population. We may imagine that the difficulty of the lower palaeolithic hunter, even if he hunted in packs, was less in finding his game than in killing it once he had come up with it. Ages

later, with the improvement of weapons, e. g., the development of the bow and arrow, the game may have been so far reduced that the difficulty was now in finding it. Under such conditions, overpopulation, in the absolute sense of the term, might occur. Unless man learned to restore the balance, either by some dimly rational limitation of his own numbers through infanticide and other checks, or by somehow conserving and increasing the game population, or unless he could migrate to regions of unexhausted food resources, the positive check of starvation would assert itself in no uncertain manner.

It is true that war may have been comparatively rare and not especially destructive of human life. It is difficult, however, to take seriously G. Elliot Smith's almost idyllic picture of the happy, carefree life of the primitive hunter—a picture which suggests the golden age of the noble savage dear to the imaginative heart of the romantic writers of the eighteenth century.[14] If primitive men did not habitually cut one another's throats, there were other checks the result of which was a high mortality. Even were we to accept Carr-Saunders' highly speculative conclusion that disease is of relatively recent origin,[15] it is obvious that the average expectation of life must have been very short. We should probably be safe in surmising that, as in the case with most animals in the wild state, few primitive adults, especially males, died a natural death. Fatigue and exhaustion were the recurrent lot of the male hunter, and frequent semistarvation the lot of all. The dangers of the chase removed many men in the prime of life. In the long sweep of human prehistory, climatic changes must have wiped out whole populations, either directly or by the forced migrations which resulted. Natural calamities, like floods and unusual extremes of seasons, changes incident to geological movements, and more recently recurring glaciation, upset the natural balance and made the life of man difficult.

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[14] Human History, 1929, chs. 5, 6.

[15] The Population Problem, 1922, pp. 155-195, 244, 245. Cf. S. H. Roberts, Population Problems of the Pacific, 1927, pp. 60, 61; R. L. Moodie, Paleopathology, an Introduction to the Study of Ancient Evidences of Disease, 1923. This monumental work covers the evidence of disease from the Cambrian on. Evidences of disease among paleolithic men are confined to apparent pathological conditions of the bony structure. While there is ample evidence of organic and other diseases among the ancient Egyptians, back to a very early date, there is practically no evidence, one way or the other, for early man. Bacteria are among the oldest known organisms, but there is at present no way of knowing when they began to cause disease in man.

Migration of the game supply, as the result either of glaciation cycles or of seasonal variations of uncertain character, meant short rations and enervating hardships. Even the Plains Indians, notwithstanding the myriads of bison which constituted their basic food supply, suffered periods of starvation when the bison herds failed to appear on schedule time.[16]

It goes without saying that infant mortality was inordinately high. In our own time, in some countries, from a fourth to a third of the infants have died before reaching one year of age. Even higher infant mortality rates were common in the Middle Ages. It is perhaps illogical to reason from the excessively high infant mortality in the crowded and unbelievably unsanitary towns of medieval Europe to rates equally high or higher among primitive hunting peoples living, uncrowded, in the open air. But what has been observed of the kind of care bestowed upon children among extant nature peoples, taken in connection with primitive man's total lack of knowledge of any healing art and of hygiene, and the absence of supplementary foods in the shape of cereal gruels,[17] warrants the conclusion that both infant and child mortality must have been incomparably high.

Nor did primitive women escape the arrows of death aimed specifically at them. There were no midwives, and such obstetrical practises as developed in the course of time were of a heroic nature. It is a strange defect of nature that a function so natural and indispensable as the preservation of the species should be attended with so much pain and danger. It is possible that the theory which attributes this maladjustment to the fact that the human race is not yet far enough along in its physical evolution to have become fully adapted to the erect posture is well founded, though if that be true, in view of the length of time man has been in existence, physiological adaptation must be an exceedingly slow process. It is known, moreover, that other animals may suffer in bringing forth young, though the human species seems to be the only one in which there is an appreciable maternal mortality. If we make allowance for the better prenatal care and the more skilful obstetrical practise among the more advanced peoples of today, there is no way of knowing whether child-bearing was more, or less, dangerous

[16] Cf. Francis Parkman, The Oregon Trail.

[17] Cf. Paul Deschamps, Comment les conditions de vie des sauvages influencent leur natalité, *Revue de l'Institut de Sociologie*, Sept., 1922, pp. 173-193.

to primitive than to modern woman, or whether physiological maladjustment has increased, or decreased, through the ages.

When we realize the conditions under which the primitive woman brought forth her offspring, however, we must infer that only women of great vitality and endurance could have gone through the experience many times. That the proportion of such women was high is probable, from the operation of selective forces generation after generation in the hard conditions of primitive life. The large amount of evidence that savage women bear children much more easily than civilized women do should probably not be taken at face value. The remarkable endurance observed among savage women in childbirth is not entirely due either to the reputed smaller size of savage infants at birth or to the fact that the physiological functions of primitive motherhood are unimpeded by the artificialities of civilization. The women of many nature peoples, among whom are mentioned various American Indian and African tribes, were laid under stringent moral obligation to utter no cry in their labor-pains. If they gave voice to pain the child would grow up a coward.[18] The long period of gestation, furthermore, must have been quite as heavy a drain on primitive woman as it is on modern, considering the character and uncertainty of the food supply and other untoward conditions of primitive woman's mode of life.[19]

THE SUPPOSED LOW FECUNDITY OF EARLY MAN

In view of these considerations with regard to the lethal forces to which the life of primitive men, women, and children was subject, and the high mortality which kept the population down to an equilibrium with available resources, it is difficult to escape the conclusion that the gross fertility of early man, during a very long time, must have been high. Otherwise, the net fertility, kept low by high mortality among infants, children, and women, would not have been high enough to maintain even a stationary population. It would seem that the relation in which the primitive hunter stood to his natural environment, for hundreds of thousands of years, gives sufficient explanation of man's practically stationary numbers down to neolithic times. Such cultural advances as took place even before the domestication of animals and the advent of agriculture would in certain circumstances enable man to strike a new and more favorable balance with resources and thereby

[18] R. Briffault, The Mothers, 1927, Vol. I, pp. 458, 459.

[19] Cf. K. Kautsky, *op. cit.*, p. 156; Briffault, *op. cit.*, p. 96.

increase his numbers. But it should not be forgotten that cultural advance also introduced preventive, and to a degree rational, checks to population growth. Such were the widespread taboos on sex intercourse in certain phases of the menstrual cycle and during lactation. Infanticide, while formally a positive check, was in effect a preventive check, since by its practise the family and the tribe could avoid more children than could be taken care of. Such limitations are of a cultural, rather than a biological, nature. If the stationary numbers and the low density of the human population down to the eve of the historical period can reasonably be attributed to the low state of culture and to high mortality, there is little reason for searching for some obscure and hypothetical biological factor, such as the low natural fecundity which Carr-Saunders argues.

Impressed by the low density and stationary numbers of primitive peoples, and by the small number of living children per family among extant savage tribes, and reasoning from the fact that the fecundity, or at least the fertility, of domesticated animals is greater than that of the same species in the wild state, Carr-Saunders risks the inference that the progressive betterment of human circumstances has occasioned an increase in fecundity as well as in net fertility.[20]

Whatever the ultimate physiological determinants of fecundity may be, its effective expression in fertility is conditioned, as Carr-Saunders points out, by three arithmetical factors: (1) the length of the reproductive period in the female, (2) the interval between births, and (3) the number of offspring at a birth. So far as the present writer knows, there is no evidence that plural births are now of more frequent occurrence than formerly. The data available with regard to the length of the reproductive period bear only indirectly on the problem here in hand. Carr-Saunders cites observations as to the difference in ages at which menstruation begins in different latitudes and diverse races.[21] It is fairly well established that puberty comes at an earlier age in the tropics than in the temperate zones, but the reproductive period also closes earlier in the tropics. It is difficult to see what light differences in the age of puberty or in the length of the child-bearing period between different races or in different climates can throw on the question of a change in fecundity in the course of human evolution. The slightly earlier age at which girls in the upper and middle classes are said by one

[20] *Op cit.*, p. 94.
[21] *Ibid.*, pp. 90-92.

authority,[22] writing over 60 years ago, to arrive at maturity may establish a distant presumption that good living conditions have a favorable influence on fecundity, but it is not established that there is any necessary correlation between early puberty and high fertility. The interval between births is influenced to so great an extent, both in savage and civilized societies, by custom and rational considerations of one sort or another, that it is by no means a certain index of fecundity.

DID PRIMITIVE MAN HAVE A MATING SEASON?

If it could be definitely established that in primitive man, as in most of the higher mammals, there is an annual oestrus cycle in the female and something in the nature of a rutting season in the male, presumption could be set up that the fecundity of primitive man was thereby less than that of modern man. Carr-Saunders is ready to believe that a cycle of this kind has been established for certain extant savages, and by inference, for primitive man. A number of writers have advanced this view. Havelock Ellis notes the statement made by physiologists from time to time that there are "annual changes in the human organism especially connected with the sex function." [23] The evidence rests on research into the reproductive processes of monkeys and certain higher apes, seasonal periodicity of births in certain lower peoples, and the widespread existence in former times of erotic festivals at certain times of the year, usually spring and autumn. Most of the higher mammals breed only once or twice a year, and among the herbivores the period is so adjusted that the young are born when food is most abundant. Except during a certain brief period the female is incapable of breeding and without sexual impulse, while the male is "either in the same condition or in a condition of latent sexuality." [24] The presence of a mating season, with sexual quiescence the rest of the year, is most definite among the herbivores. Among the carnivora it is less pronounced. It is possible that the presence of the sex cycle in animals has been over-

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[22] E. Krieger, Die Menstruation, 1869, cited by Carr-Saunders, p. 672.

[23] Studies in the Psychology of Sex, 1900, Vol. II, pp. 88 ff. Cf. E. Westermarck, History of Human Marriage, 5th ed., 1925, Vol. I, ch. 2. Perhaps the first presentation of the theory of a pairing season in early man was made by Max Kutschen, Die geschlechtliche Zuchtwahl bei den Menschen in der Urzeit, *Zeitschrift für Ethnologie, Berlin*, Vol. 8, 1876, pp. 152 ff.

[24] Havelock Ellis, *op. cit.*, p. 90.

emphasized by naturalists, who have been unduly influenced by observations on domestic herbivores.[25]

The evidence for monkeys and apes is conflicting, and as yet is not sufficient in quantity to warrant definite conclusion, but it tends strongly to establish the existence of a sexual cycle in certain species. The evidence is reviewed at length by Zuckerman,[26] who concludes that "with our present knowledge it is impossible to state with certainty whether nonpregnant mature females do or do not menstruate throughout the year," or to "judge whether or not anoestrus is experienced by any primate." [27] Hitzheimer and Heck, in Brehm's *Tierleben,* state that "in their reproductive activities monkeys and men differ from other animals in that those activities are not confined to any definite season." [28] This statement is based on the reports of field naturalists and explorers that "in every troop of monkeys young ones of every age are found at all seasons." Zuckerman, however, shows that the reports of naturalists are conflicting and of uncertain value.

> It is difficult to know how one is to arrive at the truth about primates and anoestrus from the accounts of travellers. Many of them describe breeding seasons, but their criteria vary. Some talk of rutting seasons, others of pairing seasons, while some again state that young are born only at one time of the year. Moreover, opinions about the same species sometimes conflict with each other. It is, of course, possible that the same species produces young at different times of the year in different localities, but it is hardly likely that, as has been reported, one species in some places gives birth in a particular season, while in other places it does so throughout the year.[29]

Hartman, also, points out that the evidence of travellers is unacceptable, because it is not based on carefully recorded facts.[30]

Heape made careful studies of *Semnopithecus entellus* and *Macacus rhesus* in India in 1894, and concluded it "fair to assume that there are one or more limited breeding seasons for *Semnopithecus entellus.*" In 1900 he stated without qualification that this species has a definite time for breeding. He also found strong evidence in favor of the assump-

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[25] R. Briffault, *op. cit.,* Vol. II, p. 401; L. Tillier, L'instinct sexuel chez l'homme et chez les animaux, 1889, p. 95.

[26] S. Zuckerman, The menstrual cycle in primates, *Proceedings of the Zoological Society of London,* 1930, pp. 691-754.

[27] *Ibid.,* p. 696.

[28] Brehm and Strassen, Tierleben, 1912-1920, Vol. XIII, p. 436.

[29] *Op. cit.,* p. 694.

[30] C. G. Hartman, The breeding season in monkeys, with special reference to Pithecus (Macacus) Rhesus, *Journal of Mammology,* May, 1931 (XII), p. 130.

tion that *Macacus rhesus* has one or more definite breeding seasons.[31] The strongest evidence of a breeding cycle, especially in *Macacus rhesus,* is afforded by recent work by Hartman at the Carnegie Embryological Laboratory, Baltimore. Zuckerman holds that it is "doubtful whether one may infer the reproductive habits of monkeys in the state of nature from observation of the same monkeys in captivity." The Carnegie Laboratory avoided this objection by importing, in April, 1930, a shipment direct from India of 27 females accompanied by 27 babies. After a careful technical analysis, Hartman concludes that a group of twenty-three rhesus mothers "points very neatly to October and November as near the height of the breeding season of the Bengal rhesus macaque, *Macacus rhesus,* in the state of nature." [32] Out of a total of thirty-eight rhesus conceptions of which the Laboratory thus far has record, 14 fall in November and 28 in the period October to January.[33] While Hartman's observations do not cover a large number of cases, they afford impressive confirmation of Heape's conclusion that *Macacus rhesus* has a fairly definite breeding season.

It is a far cry from rhesus to homo, however, and until more is learned of the breeding habits of the higher apes it would appear to be risky to infer a vestigial seasonal sex cycle in early man.

Many persons have thought that they observed an intensification of sexual activity among savages at certain seasons of the year, usually spring. Without inquiring into the cultural background of this periodicity, they have jumped to the easy conclusion that these peoples indicate the survival of a rutting season in early man. Westermarck uses such observations as one of the bases of his theory of the primitive pairing family, although most of his citations are to agricultural peoples far removed from the primitive hunting economy.[34] There may be a seasonal accentuation of sexual activity, as Kroeber shows for the

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[31] W. Heape, The menstruation of *Semnopithecus entellus, Philosophical Transactions of the Royal Society, London,* 1894, Vol. CLXXXV, B, pp. 411-471; The menstruation and ovulation of *Macacus rhesus, Proceedings of the Royal Society, London,* 1896, Vol. CLX, pp. 202-205; The "sexual season" of mammals and the relation of the "pro-oestrum" to menstruation, *Quarterly Journal of Microscopical Science, London,* Vol. XLIV, 1900, p. 1-70.

[32] *Op. cit.,* pp. 131, 132.

[33] *Ibid.,* p. 134.

[34] *Op. cit.*

northern California Indians,[35] but, as Briffault observes, there is a great difference between seasonal accentuation and an exclusive reproductive season.[36] How easy it is to draw untenable inferences may be seen in the case of the northern California Indians. Westermarck and Carr-Saunders quote observers to the effect that these Indians "have their rutting season as regularly as have the deer, the elk, the antelope, or any other animals," and that spring "is a literal St. Valentine's day with them, as with the natural birds and beasts of the forest."[37] This supposed sexual cycle evaporates when we turn to the work of an anthropologist who really knows the California Indians.

Speaking of the Yurok, a tribe along the lower Klamath river, Kroeber emphasizes their intense desire for wealth and the great regard they have for money (dentalium shells).

> The significant fact is that they hold a strong conviction that dentalium money and the congress of the sexes stand in a relation of inherent antithesis. This is one reason given for the summer mating season: the shells would leave the house in which conjugal desires were satisfied, and it is too cold and rainy to sleep outdoors in winter. . . . Births occurred among the Yurok and their neighbors chiefly in spring. This was, of course, not because of any animal-like impulse to rut at a certain season, as has sometimes been imagined, but because of highly specialized ideas of property and magic. . . . Since dentalia and other valuables were kept in the house, a man never slept there with his wife. . . . The institution of the sweat house rendered this easily possible. In summer, however, when the cold rains were over, the couple made their bed outdoors; with the result that it seems natural to the Yurok that children should be born in spring.[38]

The evidence is consequently far from all pointing, as Carr-Saunders thinks it does,[39] to a former condition in which conception could take place only at one season of the year.

DIET AND FECUNDITY

Two other influences may be mentioned as possible causes of induced, though hardly of inherent or racial, low fecundity in early homo. These are the influences of diet and of prolonged lactation.

It has long been known to animal breeders that proper and ample diet is favorable to fertility, but that overfeeding, tending to excessive

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[35] Handbook of the Indians of California, *Bureau of American Ethnology, Bulletin* 78, 1925, pp. 41-45.

[36] *Op. cit.*, Vol. II, p. 402, note.

[37] The quotation is from A. Johnson, The California Indians, in H. R. Schoolcraft, Historical and Statistical Information, Philadelphia, 1860, Vol. IV, p. 224.

[38] *Op. cit.*, pp. 41, 44.

[39] *Op. cit.*, pp. 94, 96, 100.

development of fat, is not.[40] Very diverse views, unsupported by scientific evidence, have been held as to the relation between diet and fertility in the human species. The general problem of diet and fertility was not put on a fully scientific basis until the existence of vitamins was discovered and their functions began to be investigated. An immense advance was made in the discovery of vitamin E and its effects, by Evans, at the University of California. Evans states that there are many relations between fertility and nutrition, some of which are as yet unconjectured.[41] It had previously been shown that deficiency in Vitamin B produced late sexual maturity and infrequent ovulations. Evans and his associates discovered that deficiency in vitamin A also caused certain characteristic abnormalities of sexual function. It was further found that "when rats are reared on some 'synthetic' food mixtures consisting of fat, carbohydrate, and protein in separate relatively pure form together with an appropriate salt mixture and the vitamins A and B, they grow well and have every appearance of health," but that "they sooner or later exhibit complete sterility."[42] We thus have abundant evidence "that sterility is a dietary deficiency disease, for it can be cured or prevented by a change in dietary regimen, a change involving the addition of certain single natural foods high in a new food factor, vitamin E, or the addition of very much smaller amounts of extracts of these foods."

Further, Evans found that the sterility disease affects males and females differently.

> In the male it eventually leads to destruction of the germ cells (eventually the entire seminiferous epithelium), but this is not the case with the female, where the ovary and ovulation are unimpaired throughout life, but where a highly characteristic disturbance occurs in gestation, namely, the death and resorption of the developing young.[43]

Other dietary deficiencies cause sterility in the female, but by interference with other steps in the reproductive process, usually by preventing oestrus, ovulation, fertilization, or implantation.

Vitamin E is present, but never highly concentrated, in a great variety of animal tissues. Muscles and fat contain several times the

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[40] F. H. A. Marshall, The Physiology of Reproduction, 2d ed., 1922, pp. 631-636.

[41] Lectures on Nutrition, given at the Mayo Foundation, 1924-1925, p. 212.

[42] *Ibid.*, p. 218.

[43] *Ibid.*, p. 220. See also Evans and Burr, The antisterility vitamin fat soluble E, *Memoirs of the University of California*, Vol. 8, 1927, ch. 3.

minimal requirement for a successful gestation [44]—an important fact in connection with the meat diet of primitive hunters. Deficiency of vitamin E cannot be argued as a probably cause of low fecundity or fertility among meat-eating peoples. The highest concentration of vitamin E is in the seeds and green leaves of certain plants, especially in the germ of wheat seed. This fact is suggestive, since early human diet, even if man was not originally a vegetarian animal, was probably supplemented by vegetable food, and, where possible, by wild cereals. Attempt to connect a hypothetical low fecundity in primitive man with dietary deficiencies (other than with those involving semi-starvation to a degree which would at times impair the individual's reproductive functions) must rest on evidence that some food element or elements, essential to successful conception, gestation, and parturition, were deficient in amount. It is possible that the numerous food taboos prevalent among nature peoples, taboos which are usually to the distinct disadvantage of the women, may have had some effect. Sometimes the taboos are so numerous that the women have a very restricted diet during pregnancy.[45] We can only speculate on the effect of primitive diet, either on fecundity or on fertility, because the nature of that diet is so uncertain.

LACTATION AND FECUNDITY

It has been a common belief that menstruation does not occur during lactation. If this were true, the widespread taboos against intercourse during lactation would in themselves have no effect on fertility. Only the prolongation of the period of nursing, in which according to this belief conception is highly unlikely to take place, would be of significance. The scientific evidence, however, while as yet not definitely conclusive, indicates that this belief has little foundation in fact. Heil, who studied 200 nursing mothers, found menstruation to be the normal thing during lactation, but less frequent in later than in earlier lactations. Fordyce reached similar conclusions, finding that menstruation occurred in 40 per cent of his cases and that in 92 per cent it returned within nine months of parturition.[46] Frank, in a later book, concludes that

> lactation may or not inhibit ovulation, ... Most women do not menstruate during the early weeks of lactation. After eight to twelve weeks a considerable number begin to menstruate. Some remain amenorrheic throughout lactation. If

[44] Lectures on Nutrition, p. 223.

[45] Briffault, *op. cit.*, p. 457.

[46] F. H. A. Marshall, The Physiology of Reproduction, 2d ed., 1922, p. 69.

lactation is unduly prolonged (1 to 2 years) permanent lactation atrophy of the uterus, accompanied by permanent amenorrhea, may supervene."[47]

Since the blood of amenorrheic lactating women has not yet been studied in relation to this problem, however, Frank considers the question still *sub judice*. So far as the evidence goes, it appears to give weight to the probability that the prolonged lactation common among primitive peoples, and indeed made necessary by the mode of their life, may have had some effect not only in keeping down fertility (this is certain), but in producing functional changes in the individual which would mean a reduced fecundity. But this, if it occurred, was a reduction of individual, and not of racial, fecundity.

THE POSITION OF WOMEN AND FECUNDITY

If we are not meticulous in distinguishing between inherent, biological, or racial reproductive power, and reproductive power as conditioned in the individual by environmental factors, it may be admitted that an increase in the average fecundity of savage women might take place with improvement in social and economic conditions, and especially in the position of woman, as was doubtless the case when hunting gave way to grazing and agriculture. Malthus attributed low fertility among savages to poor and insufficient food and "constant apprehension of being pressed by famine or by an enemy," and to "hardships and dangers of savage life which take off the attention from sexual passion," but he was not inclined to give much weight to such induced indifference as a factor influencing the number of children to a marriage. On the contrary, he attributed low fertility to the "miserable slavery" to which savage women were reduced. "This state of depression and constant labor, added to the unavoidable hardships of savage life, must be very unfavorable to the office of child-bearing; and the libertinage which generally prevails among women before marriage, with the habit of procuring abortions, must necessarily render them more unfit for bearing children afterwards," though just how Malthus does not attempt to state.[48] In other words, in Malthus' opinion, the causes of the small number of living children among savages are social and economic, not biological. Darwin believed that savages were "less prolific" than civilized peoples, and that this was due partly to the women suckling

[47] R. T. Frank, The Female Sex Hormone, 1929, pp. 54, 55, 199.

[48] T. R. Malthus, Essay on the Principle of Population, 6th ed., 1826, Vol. I, pp. 37, 39.

their infants for a prolonged period, partly to hardships and insufficient food.[49] Neither Malthus nor Darwin makes the necessary distinction between fecundity and fertility, and their views are therefore of little scientific value in the present connection. It is clear, however, that neither hardship and insufficient food—both recurringly chronic conditions of the life of early man—nor such a practise as prolonged lactation, whatever effect they may have had on fertility, or even on individual fecundity, have been shown to have any effect on inherited, racial, fecundity.

While the hardships imposed on women probably had no appreciable effect on racial fecundity, it is possible that, among the more debased savages at least, they played a not insignificant rôle in keeping down actual fertility. The evidence as to the position of women among nature peoples is somewhat conflicting. Briffault cites a large number of extant peoples among whom the women are not ill-treated, but on the contrary hold a position of "independence, influence, and even domination." On the other hand, the savage treatment of girls and women among the Australians, according to the bulk of the testimony, is almost beyond belief, and conditions in most of Melanesia appear to be little better. In parts of Africa, also, the treatment of women is exceedingly brutal.[50] It may be, however, that observers have not adequately distinguished between the kind of treatment imposed upon girls and women in the reproductive age, and the treatment of women past the menopause.

We can only guess, blindly, what woman's position among really primitive peoples in the dawn age of *Homo sapiens* was. It is possible, though not probable, that primitive humanity lived the carefree life which G. Elliot Smith depicts; it is also possible that they were savages of savages. Whether savagery is the original and natural characteristic of man, or whether it is a trait induced by thousands of years of climatic adversities, migrations, and conflicts, we do not know. About all we can venture is that however brutal the treatment of women may have been, it was not so destructive of fertility as to prevent the survival of the race, or indeed of a fertility high enough to produce that increase in numbers which enabled, and perhaps compelled, the human species to spread over the habitable earth, and, after that, high enough, on the

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[49] The Descent of Man, New York, 1871, Vol. I, p. 127.

[50] Briffault, *op. cit.*, Vol. I, pp. 311-327.

average, to maintain a fairly constant population in equilibrium with its resources.

INBREEDING AND FECUNDITY

Karl Kautsky suggests that inbreeding, due to the isolation of groups, may have had some effect in keeping down the fertility of primitive man.[51] In view of recent investigations of the effect of inbreeding on the fecundity of animals, this possibility must be regarded as remote. It was formerly thought that inbreeding is injurious in a variety of ways, among them a diminution of fecundity. In certain domestic species, inbreeding does impair fecundity, but this effect is now held to be mainly the result of confinement or of breeding highly specialized to emphasize some one character which really produces a pathological strain.[52] In recent experiments on rats, prolonged inbreeding has been accompanied by an increase in fecundity.[53] Among some peoples cousin marriages are prescribed, and no evidence appears that it adversely affects their fertility.[54] It is thus improbable that inbreeding, even to the extent that it may have been practised in primitive groups, had appreciable effect on the fecundity of the species.

THE SUPPOSED SEXUAL COLDNESS OF EARLY MAN

There remains the possibility that primitive man's fertility was relatively low because of sexual coldness, as Carr-Saunders surmises.[55] While Darwin and others have alluded to the alleged sexual indifference of savages, it is difficult to arrive at an objective judgment as to the actual prevalence of such a trait, at least in a degree sufficient to have appreciable effect on fertility. No doubt, evidences of affection between the sexes among savages and barbarians are rare. What we know as love is practically never observed. But there is a vast difference between love or affection and the raw biological urge of sex. We cannot argue sexual coldness from the apparent absence of affection. The fact that

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[51] Vermehrung und Entwicklung, 3d ed., 1921, pp. 164-166.

[52] J. A. Thomson, Heredity, 1908, p. 392; R. Briffault, *op. cit.*, Vol. I, pp. 204 ff.

[53] P. Popenoe, Experimental inbreeding, *Journal of Heredity*, 1916 (VII), pp. 74 ff.

[54] Briffault, *op. cit.*, Vol. I, pp. 218, 219. For a calculation as to the close inbreeding in certain American Indian tribes, see Clark Wissler, An Introduction to Social Anthropology, 1929, pp. 41-43. See also G. H. Lane-Fox Pitt-Rivers, The Clash of Culture and the Contact of Races, 1927, ch. 6.

[55] *Op. cit.*, p. 97.

the sexes, even husbands and wives, commonly do not live together does not prove a natural sex indifference. Any effect of an apparent sexual coldness may probably be attributed less to a biological factor than to the vagaries of cultural customs and taboos—that is, to the ruling ideas of propriety in the relations of the sexes. Be that as it may, much of the recent literature on sex in savage society is far from suggesting anything in the nature of sexual indifference. Certainly the extreme sex freedom common before marriage among most nature peoples does not suggest a constitutional sexual coldness.[56] It is possible, however, that the fatigue to which hunting and war forays subjected the men may have reduced sex urge at such times. Any physiological effect of the kind must have been small, however, compared to the restraint on sex intercourse imposed by the stringent taboos which were applied on a great variety of occasions, such as a hunting excursion or an attack projected against an enemy. It is well to remember, moreover, that sexual coldness in the male may have little effect on the birth rate, which may be the same whether intercourse is frequent or not.[57]

BIRTH STATISTICS OF PRIMITIVE PEOPLES

Finally, the reports of travellers as to the small number of children among nature peoples cannot be accepted as valid evidence. All they would see in any case would be living children. In estimating gross fertility infant mortality and abortion must be taken into account. Reliable data for some American Indian tribes indicate that there is no appreciable difference between the fertility of Indian women and that of white women. Boas found that 577 Indian women had an average of 5.9 children.[58] Hrdlička, who collected detailed family data among the Apache and Pima, found thirty-seven full-blooded Apache women, past the child-bearing age, who had had an average of 6.97 children, and thirty-five Pima women, also past the child-bearing period, with an average of 7.03 children. Because of high infant mortality, the number

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[56] On this whole matter in its cultural aspects, see Briffault, *op. cit.*, Vol. I, pp. 125, 126, 505-519.

[57] Kautsky, *op. cit.*, p. 155. Raymond Pearl (The Biology of Population Growth, 1925, ch. 8) overlooks this fact.

[58] The half-blood Indian: an anthropometric study, *Popular Science Monthly*, Oct., 1894 (XLV), pp. 761-770.

of living children was small.[59] Hrdlička has published recently, also, extensive data collected among the Oglala Sioux by Dr. James R. Walker from 1897 to 1908. The data cover 185 healthy women who married young and lived continuously in the married state during the child-bearing period. Only women who had completed that period are included. Of the total, 147 were full-blood; 38 mixed-blood (Indian-white half-breeds). Miscarriages and stillbirths were not counted. To 76 full-blood women in monogamous marriage with full-blood men, the average number of live-births was 8.1. To 48 full-blood women in polygamous marriage with full-blood men were born an average of 6.9 living children.[60] The average in various classifications of half-breed and mixed marriages was from 7.9 to 10.8. Recent intensive research among the Pueblo Indians by Miss S. B. de Aberle has found a high fertility there also.[61] Miss de Aberle collected church records of christenings, including both legitimate and illegitimate births, from 1799 to 1837 and 1892 to 1930. Data on still-births were obtained by personal interview with the Indian women. "Twenty representative informants were unanimous in saying that no contraceptive measures were used by the women of these pueblos." The women seem to want children and take great pride in their babies. The modal interval between births was found to be, for the women for whom complete and accurate data could be collected, about twenty-four months. The average number of pregnancies according to the age of the women was as follows:

	Number of individuals	*Average number of pregnancies*
Up to and including 30 years of age....	18	3.8
From 30 to 40 years of age, inclusive...	20	7.6
Over 40 years of age.....	45	9.4
Women who have stopped producing....	20	9.4
(The women in this group have had no pregnancies since October 1, 1920.)		

[59] Physiological and medical observations among the Indians of Southwestern United States and Northern Mexico, *Bureau of American Ethnology, Bulletin* 34, 1908, pp. 42, 52.

[60] Aleš Hrdlička, Fecundity in Sioux women, *American Journal of Physical Anthropology*, July-Sept., 1931 (XVI), pp. 81-90.

[61] Frequency of pregnancies and birth interval among Pueblo Indians, *American Journal of Physical Anthropology*, July-Sept., 1931 (XVI), pp. 63-80.

More careful investigations like Miss de Aberle's are needed, but the evidence collected by competent anthropologists indicates that among the American Indians at least there is no lack of fecundity. It is possible that primitive women were less fertile than the women of modern nature peoples, though thus far no real evidence has been adduced. It must be remembered that an inherent, constitutional, and racial low fecundity is a different thing from infecundity or infertility induced in the individual by ill-treatment, excessive fatigue, malnutrition, and the like. The latter is a culturally induced infecundity and is not inherent in the stock. High fertility connotes high fecundity. Further than this we cannot venture with confidence any generalization with regard to the fecundity of primitive women. It may have been just high enough to maintain the requisite fertility, or it may have been in excess of that requirement. In any case belief that early man's fecundity was lower than that of civilized man is unnecessary. It is also in conflict with the certainty that the mortality rate among primitive populations must have been very high, and that to counterbalance this, if the species were to survive, man had to have a high fertility. The stationary character and the low density of the human population during the long ages before the neolithic period and the advent of agriculture are easily explained by the hard conditions of life in the hunting economy.

BIOMETRIC STUIDES ON U. S. ARMY OFFICERS—SOMATOLOGICAL NORMS IN DISEASE*

BY LOWELL J. REED AND ALBERT G. LOVE

In the two previous papers on "Biometric Studies on U. S. Army Officers," one "Longevity in Relation to Physical Fitness"(1) and the second "Somatological Norms, Correlations, and Changes with Age"(2) we discussed the character of the basic material and its collection.

In brief, during the year 1929 we abstracted the weight, stature, chest measurement, pulse rate, systolic blood pressure, and diastolic blood pressure data, in addition to a brief of the physical and medical histories from the reports of the commission, promotion, and annual physical examinations, and from the medical records of 5,021 officers who were either in the service on January 1, 1901, or who were commissioned between that date and December 31, 1916. The maximum period of observation by means of the records extended from January 1, 1901 to approximately the middle of 1929, or 28.5 years. The average period for those on the active list was 17.5 and the total, including the ones on the retired list, 20 years. Since, however, we took the data from the reports of the physical examinations at commission of all who were in the service in January, 1901, some of whom had entered the Army many years before that time, the actual period of observation was greater.

The age distribution of the officers when originally included in the sample was given in the previous papers. Since only a few men are commissioned before the age of 21, the age grouping, 21-25, etc., is used instead of the conventional 20-24, etc., but for brevity in this study it was necessary to group by 10 years after the age 25.

The relation of disease to the physique and blood pressure is one of increasing biometric and clinical interest. One of us in a previous paper upon the subject of "Somatological Norms in Tuberculosis and

* This paper is issued jointly from the Department of Biostatistics of the School of Hygiene and Public Health of The Johns Hopkins University and the Office of the Surgeon General of the United States Army. It constitutes No. 173 in the numbered series of publications of the Department of Biostatistics.

Heart Disease"(3) discussed the frequency of those diseases in the various stature-weight and stature-chest measurement (exp.) zones of the selective service men who entered the military service during the World War. It was found in that study, as has been noted also by others, that tuberculosis is more common among the tall thin men than among the short thin ones, and many times more so than among the heavy ones, especially the short heavy ones. The build of the valvular heart cases was found to be similar. The use of the formula of the normal correlation surface to graduate the frequencies and the method of determining the percentage distribution of the disease groups in the general one was also discussed in detail in that paper.

In the present paper we shall discuss the weight-stature-age distribution of several groups of diseases.

PULMONARY TUBERCULOSIS

Pulmonary tuberculosis was much more frequent in the random sample of selective service men than it was among the more carefully selected officers. Thus the Camp Examining Boards found that of the young men in the age group 21-30, who were sent to camp, 0.53 per cent had pulmonary tuberculosis even after the local boards had rejected 1.18 per cent for that disease. In addition it caused the discharge of 0.59 per cent of them after enrollment in the military service, making a total of 2.30 per cent of cases diagnosed during a few months.

There were 106 cases found among the officers during the 87,411 years of exposure on the active list, or 0.12 per cent per year of service. Sixty-five of the 106 officers were retired as disabled and an additional 13 died while in active service, a total of 78 separations. The deaths including those among officers on the retired list numbered 47. One hundred and one of these cases are included in Table 1. The diagnosis was confirmed several years later in 78 of them at the time of retirement or death. Of the remaining 23, a few recovered, some left the service from other reasons including deaths, and some were remaining at the end of the period of observation.

Since the tuberculous cases among the selective service men which were detected upon arrival at the military camps after the hurried examinations by the local boards probably included some doubtful ones sent there for decision, the group no doubt had cases farther advanced than that of the more carefully selected officers. Consequently, we would expect that the height-weight findings of the cases detected

TABLE 1

The Cases of Pulmonary Tuberculosis Among Army Officers (Including Only Those Shown in Table 3); Number of Cases With Retirements and Deaths From Them.

AGE WHEN DIAGNOSED	21-25	26-35	36-45	46-55	TOTAL
Number	9	51	29	12	101
Age at retirement					
21-25	1	—	—	—	1
26-35	3	22	—	—	25
36-45	—	8	18	—	26
46-55	—	3	3	7	13
56-64	—	—	—	3	3
Total	4	33	21	10	68
Age at death *					
21-25	2	—	—	—	2
26-35	1	6	—	—	7
36-45	2	5	8	—	15
46-55	—	1	6	2	9
56-65	—	1	—	3	4
66-75	—	—	—	1	1
Total	5	13	14	6	38

* Twenty-eight of the deaths occurred after retirement.

among the latter would be less striking than among the former. The difference, however, is not great. Thus at 21-25, the officers in whom such a clinical diagnosis was made, either at that age or later in life, had with a stature of 70 inches a weight of 140.22 pounds, and at 26-35 one of 141.41 as compared with 136.68 among the selective men. The differences, 3.54 and 4.73 pounds are not very great when considered in relation to their probable errors, $\pm .89$ and ± 1.17.

Figure 1, which shows the percentage distribution of the cases of tuberculosis among the selective service men in the height-weight zones of their general group,(3) is reproduced here for comparison with Figures 2 and 3, which similarly show for officers at ages 26-35 and 46-55 the percentage distribution of the cases detected among them either at those ages or later in life. The three graphs demonstrate that throughout life such cases are more frequent in the area of the tall thin men, with fewer cases among the short thin ones, and still fewer among the short heavy ones. The tuberculous individual, then, has this same build not only in youth but also in old age.

TABLE 2

*Constants for Stature and Weight of the General Group of Army Officers.**

AGE	21-25	26-35	36-45	46-55	56-64
Mean:					
Stature	68.91 ± .030	69.13 ± .026	69.12 ± .026	69.05 ± .032	68.84 ± .055
Weight	146.66 ± .23	154.49 ± .24	160.19 ± .26	164.02 ± .33	164.91 ± .58
Weight at 70 inches	151.27 ± .19	158.21 ± .21	164.10 ± .23	168.19 ± .30	169.81 ± .53
Stand. Dev.:					
Stature	2.17 ± .021	2.16 ± .018	2.18 ± .018	2.19 ± .023	2.21 ± .039
Weight	16.57 ± .16	20.18 ± .17	21.87 ± .18	22.55 ± .24	22.93 ± .41
Coeff. of Var.:					
Stature	3.15 ± .031	3.12 ± .026	3.16 ± .026	3.17 ± .033	3.21 ± .057
Weight	11.30 ± .11	13.06 ± .11	13.65 ± .12	13.75 ± .15	13.91 ± .25
Correlation	.5536 ±.0096	.4558 ±.0094	.4429 ±.0095	.4280 ±.0121	.4059 ±.0212
Number	2,360	3,198	3,232	2,071	720

* For comparison with data in Tables 3, 5, 7, 10 and 11.

Furthermore, as life insurance medical directors have pointed out, the weight of those who ultimately develop tuberculosis is below the average many years before the disease becomes evident clinically. Thus in the words of Huntington in 1876(4): "Consumption very, very, often does send out its warning voice far in advance of its active coming, and in many cases this interference with the tissue-making powers of the human system foreshadows the coming of tuberculosis." The officers at the age 21-25, who developed this disease 20 years later, had a weight of 138.39 pounds with a stature of 70 inches as compared with 151.27 in the general group, a difference of 12.88 pounds, which is certainly a significant one. Again at the age 26-35, those who develop the disease 10 years later had a weight of 142.95 with a stature of 70 inches as compared with 158.21 in the general group, a difference of 15.26 pounds. The data in Table 3 show that at every age those who

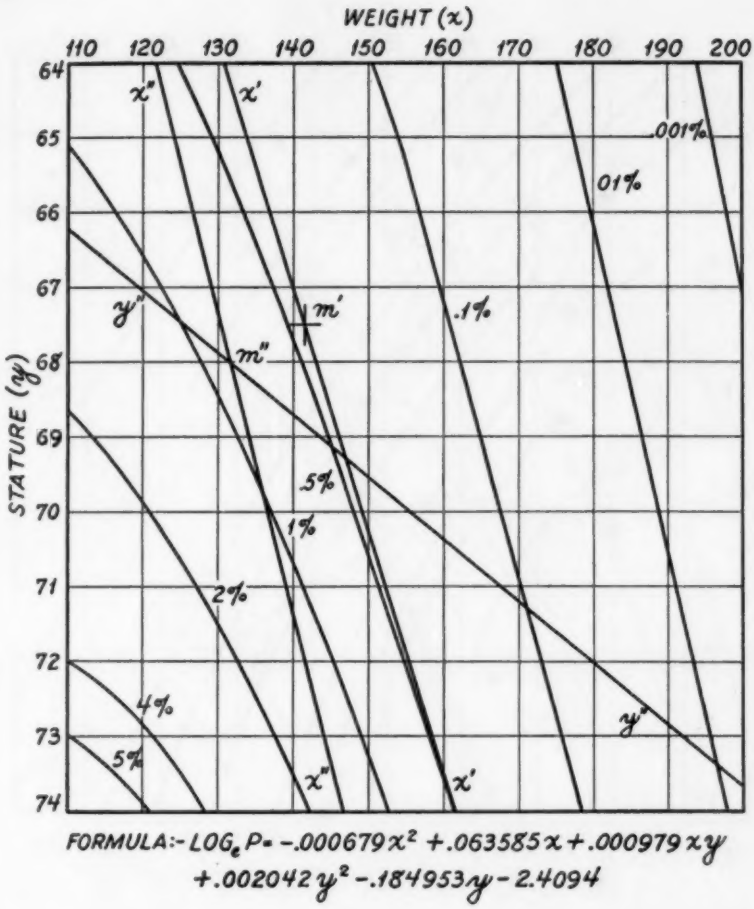

FIG. 1. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 21-30 OF PULMONARY TUBERCULOSIS CASES WHICH WERE DIAGNOSED AMONG SELECTIVE SERVICE MEN. AVERAGE, 0.54%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

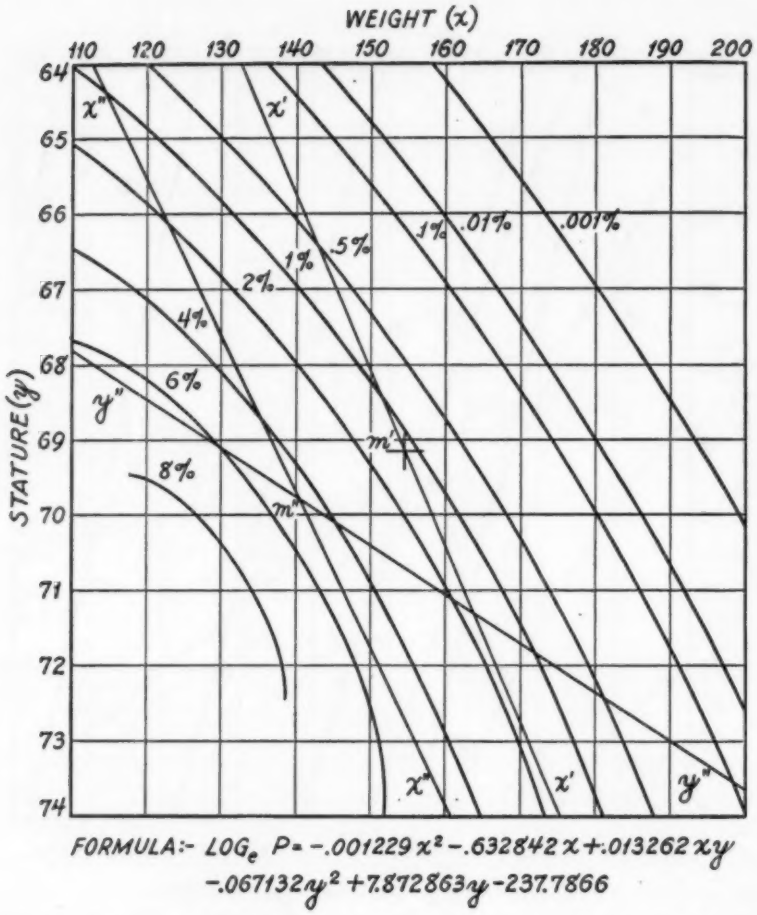

FIG. 2. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 26-35 OF PULMONARY TUBERCULOSIS CASES WHICH WERE DIAGNOSED AT THAT AGE OR LATER IN LIFE. THE AVERAGE WAS 1.91%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

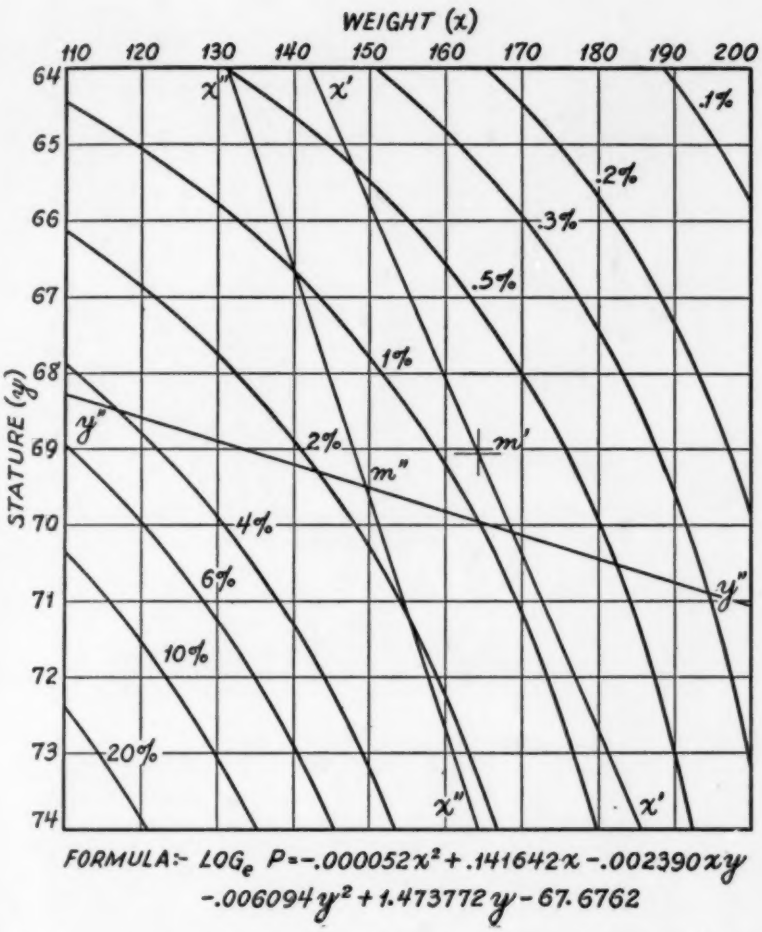

FIG. 3. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 46-55 OF PULMONARY TUBERCULOSIS CASES WHICH WERE DIAGNOSED AT THAT AGE OR LATER IN LIFE. THE AVERAGE WAS 1.06%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

TABLE 3

*Constants for Stature and Weight of Army Officers Who had Pulmonary Tuberculosis Either at the Age When Weighed and Measured or Later in Life.**

DATA AT AGE	21-25				26-35			36-45		46-55
Disease:	At age	7.5 yrs. later	20 yrs. later	At age or later	At age	10 yrs. later	At age or later	At age	At age or later	At age or later
Mean:										
Stature	69.90 ± .57	69.07 ± .25	68.93 ± .36	69.12 ± .20	69.65 ± .14	69.78 ± .25	69.79 ± .16	69.50 ± .25	69.59 ± .18	69.50 ± .29
Weight	139.80 ± 2.61	136.73 ± 1.56	134.57 ± 2.18	136.62 ± 1.20	138.21 ± 1.70	142.22 ± 1.64	140.39 ± 1.40	144.46 ± 1.94	144.78 ± 1.55	149.41 ± 3.03
Weight at 70 inches	140.25 ± .99	140.53 ± 1.17	138.39 ± 1.77	140.22 ± .87	139.73 ± 1.46	142.95 ± 1.44	141.41 ± 1.16	145.57 ± 1.86	146.42 ± 1.37	151.03 ± 2.87
Stand. Dev.:										
Stature	2.66 ± .40	2.03 ± .18	1.98 ± .25	2.13 ± .14	1.93 ± .14	1.74 ± .17	1.89 ± .12	1.93 ± .18	1.95 ± .13	2.04 ± .21
Weight	12.25 ± 1.85	12.65 ± 1.10	12.12 ± 1.54	12.55 ± .85	16.52 ± 1.20	11.66 ± 1.16	16.16 ± .99	14.70 ± 1.37	16.44 ± 1.91	21.04 ± 2.14
Coeff. of Var.:										
Stature	3.81 ± .58	2.94 ± .26	2.87 ± .37	3.09 ± .21	2.77 ± .20	2.50 ± .25	2.71 ± .17	2.77 ± .26	2.81 ± .19	2.93 ± .30
Weight	8.77 ± 1.33	9.25 ± .81	9.00 ± 1.16	9.19 ± .62	11.95 ± .88	8.20 ± .82	11.51 ± .71	10.17 ± .96	11.35 ± .77	14.09 ± 1.46
Correlation	.9250 ±.0308	.6579 ±.0700	.5851 ±.1185	.6913 ±.0498	.5068 ±.0764	.4813 ±.1081	.5577 ±.0596	.2975 ±.1206	.4744 ±.0732	.3172 ±.1293
Number	10	30	14	54	43	18	61	26	51	22

* See Table 2 for comparative data of the general group.

ultimately develop pulmonary tuberculosis had the same characteristic tall spare build, and that it is apparent from 7.5 to 20 years before the disease becomes clinically evident.

Not only is the mean weight of the tuberculous group at each age much below that of the general one, but the variability of it as shown both by the standard deviation and the coefficient of variability is considerably less, indicating that although some of the patients have a normal build, the general run of them is below the average. This smaller dispersion was also apparent among the selective service men for whom the standard deviation of the diseased group was 14.95±.10 pounds as compared with 17.42±.0089 in the general one; and the coefficient of variability in the former was 11.35 as compared with 12.31 in the latter.

The tuberculous selective service men, as well as the officers at every age, had a mean stature greater than that of the general groups, but the variability of it was about the same.

CARDIOVASCULAR-RENAL DISEASES

Cardiovascular-renal diseases caused 396, or 36.8 per cent, of the 1,076 deaths among the 5,021 officers during this experience. The age 21-90 death rate among them from this group of diseases was 3.48 per 1,000 years of life as compared with 3.65 among the white males of the same ages in the registration area during 1919-1921.(1) The group was also responsible for 218, or 24.7 per cent, of the 881 retirements for disability before age 64, the retirement rate from it being 2.49 per 1,000 years of active service as compared with 2.32 from nervous and mental diseases and 0.85 from tuberculosis of all forms. The serious character of the cardiovascular-renal diseases is shown by the fact that after retirement the expectancy of life at every age was only one-half as great as that of the general group.

We have divided this group into three sub-groups: 1. The valvular diseases of early life; 2. the degenerative diseases of later life; 3. the syphilitic manifestations. The last group was a small one, and will not be considered in this discussion.

Valvular Heart Disease (other than aortic regurgitation)

This group includes the cases which occur as the result of acute rheumatic fever and other infectious diseases during the first 20 or 25 years. Less than 1.0 per cent of the officers weighed or measured at any age had such a disease diagnosed then or later in life. Such cases

* See Table 2 for comparative data of the general group.

as were reported probably either escaped detection, or were considered functional conditions, when the officers were commissioned. During this experience there were 65 cases diagnosed as compared with 106 of pulmonary tuberculosis. Forty-one of the officers were retired as disabled and an additional 7 died, making a total of 48 with a disability and fatality rate of 74 per cent. The deaths at all ages among both active and retired officers amounted to 16. Fifty-nine of the 65 cases are included in Table 4. In 40, or 68 per cent, the diagnosis was confirmed later during this experience at the time of retirement or death.

TABLE 4

Cases of Valvular Heart Diseases (Other Than Aortic Regurgitation) Among Army Officers (Including Only the Cases Shown in Table 5); Number of Cases With Retirements and Deaths From Them.

AGE WHEN DIAGNOSED	21-25	26-35	36-45	46-55	56-64	TOTAL
Number	1	11	19	18	10	59
Age at retirement						
21-25	—	—	—	—	—	—
26-35	—	7	—	—	—	7
36-45	1	3	7	—	—	11
46-55	—	—	3	10	—	13
56-64	—	—	—	1	4	5
Total	1	10	10	11	4	36
Age at death *						
26-35	—	—	—	—	—	—
36-45	—	—	1	—	—	1
46-55	—	—	1	3	—	4
56-65	—	—	—	—	1	1
66-75	—	—	—	—	1	1
Total	—	—	2	3	2	7

* Three of the deaths occurred among the retired officers.

The group is of special interest on account of the persistence of the build of the patients. In the paper to which reference has been made,(3) it was shown that the valvular heart diseases, as detected among the selective service men between the ages of 21 and 30, like tuberculosis were much more common among the thin individuals and especially the tall thin ones. With reference to the character of cases, the data show that of the 6,687 cases detected at camps among the

870,000 men, 92 per cent of diseases with a specified diagnosis had lesions of the mitral valve; and practically all of them were the result of infectious diseases which had occurred early in life(3).

TABLE 5

*Constants for Stature and Weight of Army Officers Who had a Valvular Heart Disease (Other Than Aortic Regurgitation) Either at the Age When Weighed and Measured or Later in Life.**

DATA AT AGE	21-25	26-35	36-45		56-64
Disease:	At age or later	At age or later	At age	At age or later	At age
Mean:					
Stature	69.33 ± .39	69.76 ± .28	69.89 ± .33	69.47 ± .24	69.60 ± .42
Weight	145.06 ± 2.30	149.83 ± 1.96	151.84 ± 3.06	156.19 ± 2.34	163.20 ± 3.33
Weight at 70 inches	145.84 ± 2.25	150.44 ± 1.83	152.31 ± 2.72	158.75 ± 2.04	163.50 ± 3.32
Stand. Dev.:					
Stature	2.47 ± .28	2.21 ± .20	2.12 ± .23	2.10 ± .17	1.96 ± .30
Weight	14.45 ± 1.62	15.67 ± 1.39	19.76 ± 2.16	20.78 ± 1.65	15.63 ± 2.36
Coeff. of Var.:					
Stature	3.57 ± .40	3.16 ± .28	3.04 ± .33	3.02 ± .24	2.82 ± .42
Weight	9.96 ± 1.13	10.46 ± .94	13.02 ± 1.45	13.31 ± 1.08	9.57 ± 1.46
Correlation	.1985 ±.1486	.3557 ±.1094	.4583 ±.1222	.4870 ±.0587	.0941 ±.2114
Number	18	29	19	36	10

* See Table 2 for comparative data of the general group.

Table 5 gives the height and weight constants for such cases in the officer group. The mean weight of the officer patients was about the same as that of the selective service men of the same age. Thus at 21-25, the officers in whose cases such a clinical diagnosis was made either then or later had a weight of 145.84 pounds with a stature of 70 inches as compared with 144.79 for the selective service men, ages 21-30. The

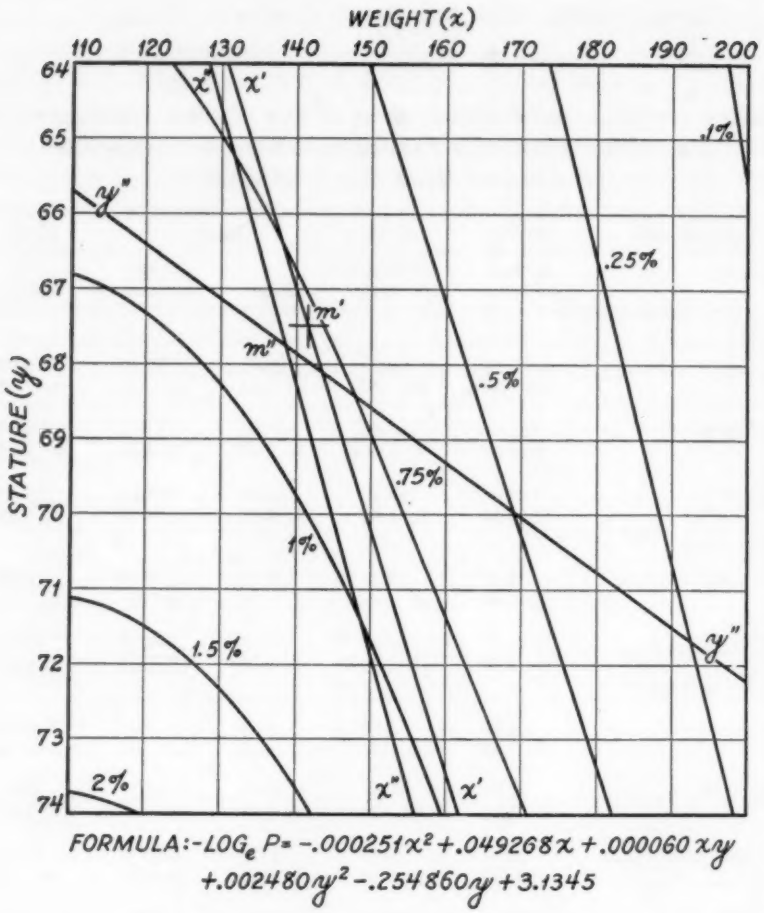

FIG. 4. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 21-30 OF VALVULAR HEART DISEASES (EXCL. AORTIC CASES) DIAGNOSED AMONG SELECTIVE SERVICE MEN. AVERAGE, .78%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

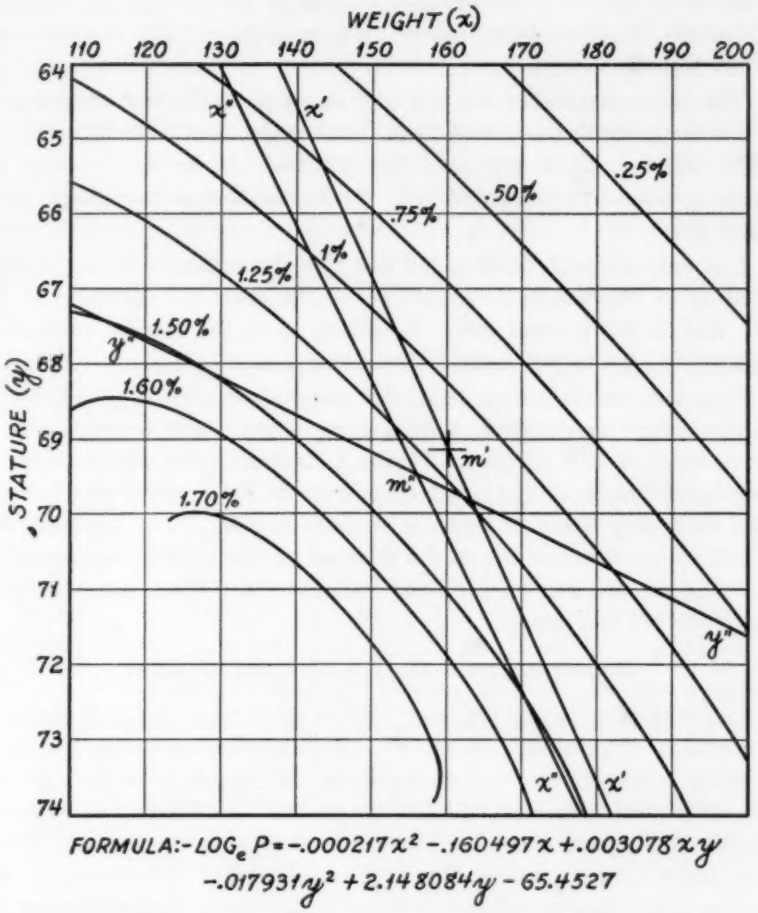

FIG. 5. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 36-45 OF VALVULAR HEART DISEASES (EXCL. AORTIC CASES) DIAGNOSED AT THAT AGE OR LATER IN LIFE. AVERAGE, 1.11%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

difference of 1.05 pounds is less than its probable error ±2.30. An examination of the table shows that at every age the weight with 70 inches of stature is much lower than that of the general group. The differences by ages (exc. 46-55) are —5.43, —7.77, —11.79, and —6.31 pounds.

The mean stature of the valvular cases is uniformly greater than that of the general group, and the differences by ages from the youngest to the oldest (exc. 46-55) .42, .63, .77, and .76 inches, although not always great when compared with their probable errors, seem to be significant.

Not only are such patients tall and thin throughout life, but the distribution of the weight as measured by the standard deviation is less than that of the general body, indicating as in the case of pulmonary tuberculosis, the compactness of the group.

Figure 4, which is reproduced for comparison with Figure 5, shows the percentage distribution of such cases in the height-weight zones of the selective service men(3). Figure 5 similarly gives the grouping in the general height-weight zones of officers at the ages of 36-45 of the cases diagnosed either at that age or later in life. It is apparent that the percentage distributions of the diseased groups on the two graphs are very similar, and that on each one such cases are much more frequent among the tall thin men.

Degenerative Cardiovascular-Renal Diseases

We have classified as the degenerative cardiovascular-renal diseases: myocarditis, arteriosclerosis, arterial hypertension, nephritis, apoplexy, coronary sclerosis, coronary thrombosis, and angina pectoris. During this period, which included the last 1.5 years of the Philippine Insurrection, the subsequent guerrilla warfare in that country, and the World War, these diseases caused the death or disability retirement, of five times as many men as military combat. They were responsible for the retirement or death on the active list of 254 officers, or 19 per cent of those from all causes. Including the deaths which occurred after retirement, they caused 35 per cent of the total, and were responsible for twice as many as cancer, tuberculosis, and military combat all combined. During this experience 579 officers received treatment in hospital for one of these diseases. Of this number, 167 were retired as disabled and an additional 297 died, making a total of disabilities or deaths of 464, or 80 per cent of all cases.

Of the 579 cases, 429 are included in Table 6. Some of this number left the Army and some died or retired on account of other causes. Of the 429 cases, 152, or 35 per cent, were retired as physically disabled from this cause; and 223 (including 53 of the 152 retired), or 52 per cent, died as the result of these diseases during this experience, which comprised an average of 17.5 years on the active list and of life of 20. One hundred and seventy of the deaths occurred among officers who had not been retired as disabled from this cause. These deaths added to the 152 retirements make a total of 322, or 75 per cent of the 429 cases, which is sufficient evidence of the gravity of the diseases as diagnosed.

TABLE 6

Cases of Degenerative Cardiovascular-Renal Diseases Among Army Officers (Including Only the Cases Shown in Tables 7, 8, 9); Number of Cases With the Retirements and Deaths From Them.

AGE WHEN DIAGNOSED	26-35	36-45	46-55	56-64	TOTAL
Number	30	74	162	163	429
Age at retirement:					
26-35	7	—	—	—	7
36-45	3	27	—	—	30
46-55	2	6	54	—	62
56-64	—	1	12	40	53
Total	12	34	66	40	152
Age at death:					
26-35	7	—	—	—	7
36-45	3	13	—	—	16
46-55	2	8	41	—	51
56-65	—	3	16	58	77
66-75	—	1	20	37	58
76+	—	—	2	12	14
Total	12	25	79	107	223

NOTE—Fifty-three of the deaths occurred among the 152 retired officers.

Height-weight.—Table 7, which gives the height and weight constants in each age group of the officers in whose case a clinical diagnosis of one of these diseases was made either at that age or later, shows that there is a very material difference between the mean weight of this group and that of either the valvular disease or the tuberculous one. In

the two last named groups the weight was appreciably less than that of the general group, but in this one, and especially after the age of 26-35, the mean of the disease group is much greater than the one of the general group.

In our paper "Somatological Norms, Correlations, and Changes with Age"(2) we discussed the material increase in the mean weight with the advance in age and found that the greatest amount of it occurred during the thirties and forties with but little, if any, after fifty-five. This increase we called physiological because it antedates the degenerative diseases with which it is often associated later in life. We did not intend, however, to imply that this increase was desirable or that it was harmless.

At 21-25, there was no material difference between the mean weight of those who later develop a cardiovascular-renal disease and that of the general group. Similarly at 26-35, there was no real difference between the weight of the general group and the one of those who developed the disease at that age, but there was a significant increase in it among those who developed it 10 or 20 years later. Beginning with age 36-45, there was a real difference at every age between the average weight of the general group and that of the cases diagnosed either then, or 10 or 20 years later. Thus the officers who showed the first clinical evidence of this degenerative type of disease during the decade from 56-64 weighed at that time 177.47 pounds with a stature of 70 inches, 10 years earlier, 178.91 pounds, and 20 years earlier 177.46, as compared with 169.81, 168.19 and 164.10 for the general group. Similarly, the ones who died between the ages 66 and 75 as a result of one of these diseases had at 56-64 a weight with a stature of 70 inches of 181.05, and at 46-55 one of 183.64 as compared with 169.81 and 168.19, respectively, for the general group.

The mean stature of the officers with such a disease is uniformly lower than the one of the general group. Although in the majority of instances it is not significantly so when tested with its probable error, the persistence of the difference apparently indicates a real one.

Of the men in the general group at the age of 26-35, 4.25 per cent either at that age or within 20 years had a disease of this kind. Figure 6 shows, in the height-weight zones of the general group at that age, the percentage distribution of these cases which were diagnosed either then or later in life. Although such diseases occur among light-weight men, the percentage is quite low. The ones who had an average weight

TABLE 7

TABLE 7

*Constants for Stature and Weight of Army Officers Who had a Degenerative Cardiovascular-Renal Disease Either at the Age When Weighed and Measured or Later in Life.**

DATA AT AGE	21-25				26-35				36-45	
Disease:	7.5 yrs. later	17.5 yrs. later	27.5 yrs. later	7.5 yrs. or later	At age	10 yrs. later	20 yrs. later	At age or later	At age	10 yrs. later
Mean:										
Stature	68.50 ± .28	69.06 ± .29	68.65 ± .27	68.63 ± .17	68.33 ± .38	69.14 ± .20	69.06 ± .19	68.96 ± .13	69.22 ± .18	69.04 ± .16
Weight	143.28 ± 2.74	144.16 ± 2.29	144.15 ± 2.16	143.20 ± 1.30	147.76 ± 2.36	161.74 ± 2.61	166.78 ± 2.20	161.34 ± 1.52	168.26 ± 2.21	174.41 ± 1.72
Weight at 70 inches	155.02 ± 1.68	148.12 ± 1.93	151.04 ± 1.67	149.82 ± 1.02	152.56 ± 2.05	166.29 ± 2.37	170.20 ± 2.08	166.68 ± 1.36	173.56 ± 1.85	179.69 ± 1.49
Stand. Dev.:										
Stature	1.74 ± .20	2.42 ± .21	2.32 ± .19	2.35 ± .12	2.55 ± .27	1.98 ± .14	2.37 ± .14	2.27 ± .09	2.11 ± .13	2.47 ± .11
Weight	17.24 ± 1.94	18.92 ± 1.62	18.70 ± 1.53	18.42 ± .92	16.01 ± 1.67	25.37 ± 1.85	26.87 ± 1.55	26.22 ± 1.07	26.18 ± 1.56	26.58 ± 1.22
Coeff. of Var.:										
Stature	2.54 ± .29	3.51 ± .30	3.39 ± .28	3.42 ± .17	3.73 ± .39	2.87 ± .21	3.43 ± .20	3.30 ± .14	3.04 ± .18	3.57 ± .16
Weight	12.03 ± 1.37	13.12 ± 1.14	12.97 ± 1.08	12.86 ± .65	10.84 ± 1.14	15.69 ± 1.17	16.11 ± .96	16.25 ± .68	15.56 ± .95	15.24 ± .72
Correlation	.7898 ±.0598	.5418 ±.0856	.6373 ±.0687	.6145 ±.0438	.4588 ±.1308	.4162 ±.0850	.3219 ±.0733	.4433 ±.0465	.5463 ±.0591	.5068 ±.0329
Number	18	31	34	92	21	43	68	136	64	108

TABLE 7—*Continued*

DATA AT AGE	36-45 (cont.)		46-55				56-64			
Disease:	20 yrs. later	At age or later	At age	10 yrs. later	20 yrs. later	At age or later	At age	10 yrs. later	20 yrs. later	At age or later
Mean:										
Stature	68.84 ± .22	68.94 ± .11	69.20 ± .15	68.85 ± .15	68.41 ± .27	68.98 ± .10	68.75 ± .15	67.74 ± .26	69.23 ± .46	68.59 ± .13
Weight	167.09 ± 2.78	171.45 ± 1.22	172.66 ± 1.64	173.53 ± 1.70	166.82 ± 4.00	172.34 ± 1.14	170.03 ± 1.62	162.77 ± 2.97	171.46 ± 4.67	169.33 ± 1.40
Weight at 70 inches	177.46 ± 1.93	177.76 ± 1.04	175.07 ± 1.58	178.91 ± 1.39	183.64 ± 2.79	177.34 ± 1.03	177.47 ± 1.34	181.05 ± 2.46	175.93 ± 3.82	178.20 ± 1.14
Stand. Dev.:										
Stature	2.22 ± .16	2.31 ± .07	2.36 ± .10	2.25 ± .11	1.65 ± .19	2.24 ± .07	2.26 ± .11	2.08 ± .18	2.45 ± .32	2.30 ± .09
Weight	27.62 ± 1.96	26.60 ± .86	26.43 ± 1.14	25.14 ± 1.20	24.43 ± 2.83	25.84 ± .80	24.14 ± 1.15	24.09 ± 2.10	24.98 ± 3.30	24.94 ± .99
Coeff. of Var.:										
Stature	3.23 ± .23	3.36 ± .11	3.41 ± .15	3.26 ± .16	2.41 ± .28	3.25 ± .10	3.29 ± .16	2.96 ± .26	3.55 ± .47	3.35 ± .13
Weight	16.53 ± 1.21	15.51 ± .51	15.31 ± .69	14.49 ± .71	14.65 ± 1.73	14.99 ± .48	14.20 ± .69	14.80 ± 1.32	14.57 ± 1.97	14.73 ± .60
Correlation	.7186 ±.0486	.5219 ±.0333	.2686 ±.0576	.5734 ±.0453	.7151 ±.0799	.4225 ±.0361	.5587 ±.0462	.6990 ±.0846	.5753 ±.1252	.5801 ±.0373
Number	45	217	118	100	17	235	101	30	13	144

* See Table 2 for comparative data of the general group.

Fig. 6 ... group ... point ...

STATURE (Y)

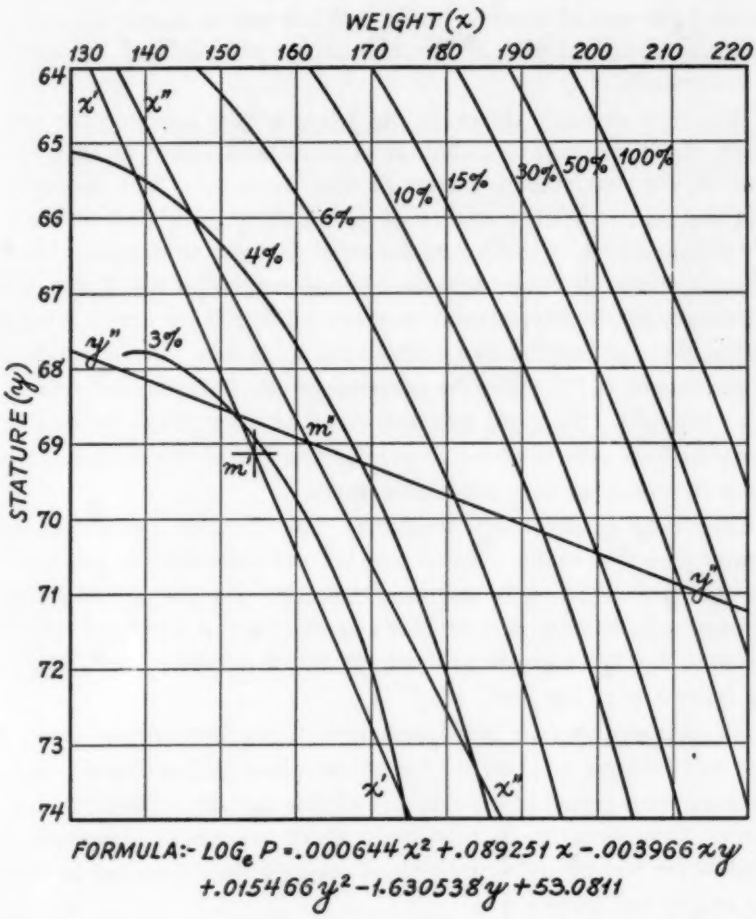

FIG. 6. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 26-35 OF DEGENERATIVE CARDIOVASCULAR-RENAL DISEASES DIAGNOSED AT THAT AGE OR LATER IN LIFE. AVERAGE, 4.25%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

Number 45 217 118 100 17 235 101 30 13 144

* See Table 2 for comparative data of the general group.

had a disease expectancy of less than 3 per cent; and those who were 15 lbs. above the average about 4.5 per cent. With a stature of 70 inches 3 per cent of those weighing 158 had such a disease then or later in life as compared with 10 per cent at 192 pounds; and 100 per cent at 222 pounds.

Figure 7 similarly shows, in the height-weight zones of the men at 46-55, the percentage distribution of ones who either at that age or later in life developed a disease of this character. The average was 11.4 per cent. With a stature of 70 inches, 7.5 per cent of the ones at 156 pounds had a cardiovascular-renal disease; as compared with 75 per cent at 230. Officers with an average weight for age had a disease expectancy of about 9 per cent, as compared with 8 per cent for the ones who by diet and exercise had retained the 26-35 one. On the other hand an increase of 15 lbs. above the age average raised the disease expectancy to 13 per cent. Since the probable error of distribution in the disease group in both ages is rather large, the position of the percentage lines naturally varies within considerable limits.

Not only is the mean weight of this diseased group materially greater than that of the general one, but the variability as measured by the standard deviation is also much greater. At 46-55, two-thirds of the men who had the disease either at that age or developed it within 20 years, weighed between 146 and 198 lbs., the disease percentage varying from 2 to 22 per cent.

Weight and systolic blood pressure.—Since the systolic blood presure was first recorded on the reports of physical examination in 1916, the maximum period of observation of this variable in any group is 14 years. Consequently, in this study there are fewer observations in Table 8 for weight and systolic blood pressure than there are in Table 7 for weight and stature.

As stated in "Somatological Norms, Correlations, and Changes with Age"(2) there is but little increase in the mean of the systolic blood pressure until after 45 years of age, and 50 per cent of the total increase occurs between 56 and 64. We called this a pathological increase because it occurs during the period when the degenerative changes are taking place in the cardiovascular-renal system. We showed also that when we excluded the known diseased cases, the means from 21-25 to 56-64 were 122.04, 123.61, 125.29, 129.15, and 136.58.

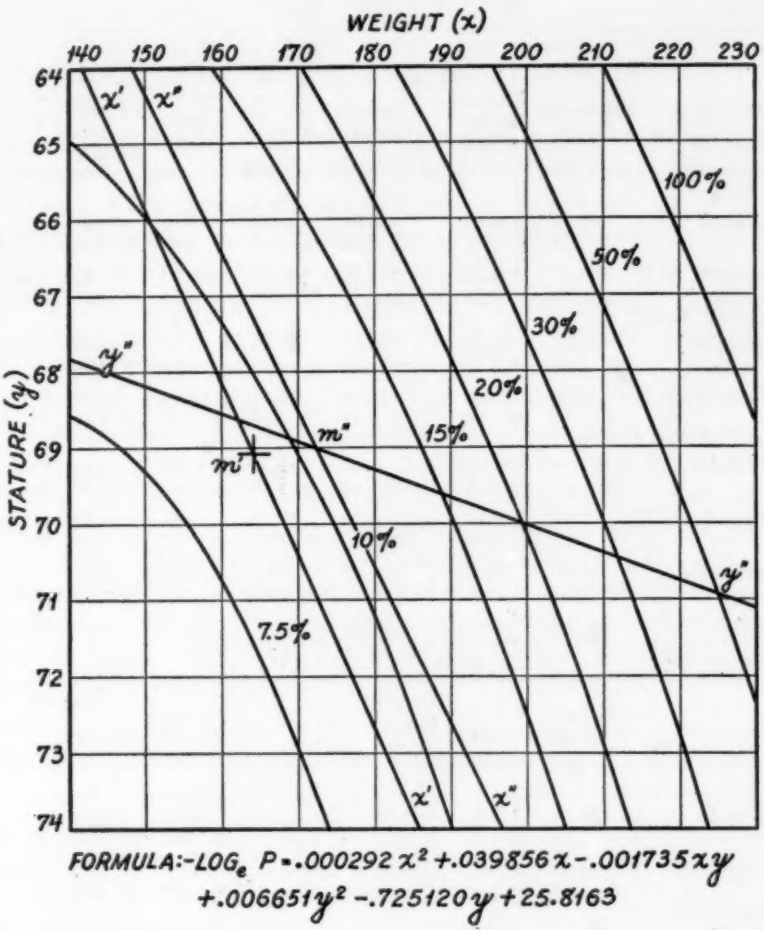

FIG. 7. DISTRIBUTION IN THE STATURE-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 46-55 OF DEGENERATIVE CARDIOVASCULAR-RENAL DISEASES DIAGNOSED AT THAT AGE OR LATER IN LIFE. AVERAGE, 11.35%.

The x' and x'' lines are weight on stature regressions of the general and disease groups; y'' the stature on weight regression of the latter. m' and m'' are the mean points of stature and weight.

TABLE 8

Constants for Weight and Systolic Blood Pressure of the General Group of Army Officers and Also of Those Who had a Degenerative Cardiovascular-Renal Disease Either at the Age When Weighed and Measured or Later in Life.

DATA AT AGE	26-35		36-45				
				Disease			
	General Group	Disease at age or later	General Group	At age	10 yrs. later	More than 10 yrs. later	At age or later
Mean:							
Weight	154.45 ± .32	159.22 ± 3.87	159.64 ± .28	171.28 ± 3.01	175.45 ± 2.74	173.91 ± 4.93	174.38 ± 1.89
Systolic B. P.	123.78 ± .16	133.33 ± 2.39	125.91 ± .15	140.59 ± 2.80	140.30 ± 1.69	126.45 ± 2.99	140.06 ± 1.44
Standard Dev.:							
Weight	19.61 ± .24	29.83 ± 2.74	20.85 ± .20	27.86 ± 2.13	29.57 ± 1.94	24.26 ± 3.49	28.51 ± 1.34
Systolic B. P.	9.28 ± .11	18.41 ± 1.69	11.00 ± .11	25.91 ± 1.98	18.25 ± 1.20	14.70 ± 2.11	21.69 ± 1.02
Coeff. of Var.:							
Weight	12.70 ± .15	18.74 ± 1.78	13.06 ± .14	16.27 ± 1.28	16.85 ± 1.13	13.95 ± 1.90	16.35 ± .79
Systolic B. P.	7.50 ± .091	13.81 ± 1.29	8.74 ± .085	18.43 ± 1.45	13.01 ± .87	11.63 ± 1.70	15.49 ± .75
Correlation ...	.2427 ±.0160	.2643 ±.1207	.2507 ±.0127	.1831 ±.1044	.3965 ±.0781	.4047 ±.1701	.2948 ±.0607
Number	1,576	27	2,463	39	53	11	103

DATA AT AGE	46-55				56-64		
		Disease				Disease	
	General Group	At age	10 yrs. later	At age or later	General Group	At age	At age or later
Mean:							
Weight	163.73 ± .35	172.83 ± 1.76	171.88 ± 2.14	171.17 ± 1.35	164.84 ± .66	168.73 ± 1.91	167.86 ± 1.72
Systolic B. P.	130.90 ± .23	150.45 ± 1.46	144.66 ± 1.86	148.62 ± 1.15	139.77 ± .57	157.88 ± 2.14	156.48 ± 1.92
Standard Dev.:							
Weight	21.53 ± .25	25.57 ± 1.24	24.16 ± 1.51	25.08 ± .95	22.64 ± .46	24.54 ± 1.35	23.72 ± 1.21
Systolic B. P.	14.32 ± .16	21.16 ± 1.03	21.02 ± 1.32	21.36 ± .81	19.70 ± .40	27.49 ± 1.51	26.49 ± 1.35
Coeff. of Var.:							
Weight	13.15 ± .15	14.79 ± .74	14.06 ± .90	14.65 ± .57	13.73 ± .29	14.54 ± .82	14.13 ± .74
Systolic B. P.	10.94 ± .13	14.06 ± .70	14.53 ± .93	14.37 ± .56	14.09 ± .29	17.41 ± .99	16.93 ± .89
Correlation ...	.1725 ±.1057	.0273 ±.0688	.2240 ±.0841	.1163 ±.0531	.1470 ±.0283	.0067 ±.0779	.0259 ±.0723
Number	1,747	96	58	157	543	75	87

The percentage of diseased cases in the general group increased from 1.7 at age 26-35 to 4.1 in the next one, then to 8.9, and finally to 16.4 in the 56-64 one. At 26-35, the systolic reading of the officers who had a cardiovascular-renal disease either at that age or later in life was 9.55 mm. greater than that of the general group. This difference increased to 14.15 mm. during the next age period, then to 17.72, after which there was a slight decline to 16.71 at 56-64. If, however, we compare the mean of the diseased groups with that of the general ones after excluding the known pathological cases, the differences are 9.71, 14.77, 19.47, and 19.90.

We have found that these patients had the characteristic increase in weight as much as 20 years before the disease became clinically apparent. The increase in the systolic blood pressure, however, did not become apparent until 10 years, or even less, before the clinical diagnosis was made. Thus at 36-45, there was no increase in it among the officers who developed a disease of this character within the maximum limits of this period of observation, which was about 14 years; but it was as high for those who developed one 10 years later as it was for the cases diagnosed at that same age. At 46-55, however, the mean for those who had such a disease at that age was 6 mm. greater than the one for the cases diagnosed 10 years later. Furthermore in all instances the mean of the pathological group continued to increase until the age when the diseases became manifest clinically.

In the general group, weight has a rather small positive correlation with systolic blood pressure which is not increased by the elimination of stature by the partial correlation method. In the cardiovascular-renal group, this correlation is slightly greater at 26-35 and 36-45, but it is considerably less at 46-55, and much lower at 56-64 when it is so small as to be insignificant. To test still further the effect of stature upon this coefficient, the systolic of the disease group at 56-64 was correlated with weight using as an assumed mean the regression weight, thus varying it for every inch in stature. The coefficient thus obtained was .0408 as compared with .0259 in Table 8.

Figures 8 and 9 show the percentage distribution in the various weight-systolic zones of the general group at ages 46-55 and 56-64 of the cases who had clinical evidence of one of these diseases at the age in question or later in life. At 46-55, 8.9 per cent of the officers and at 56-64, 16.0 per cent of them had such a diagnosis.

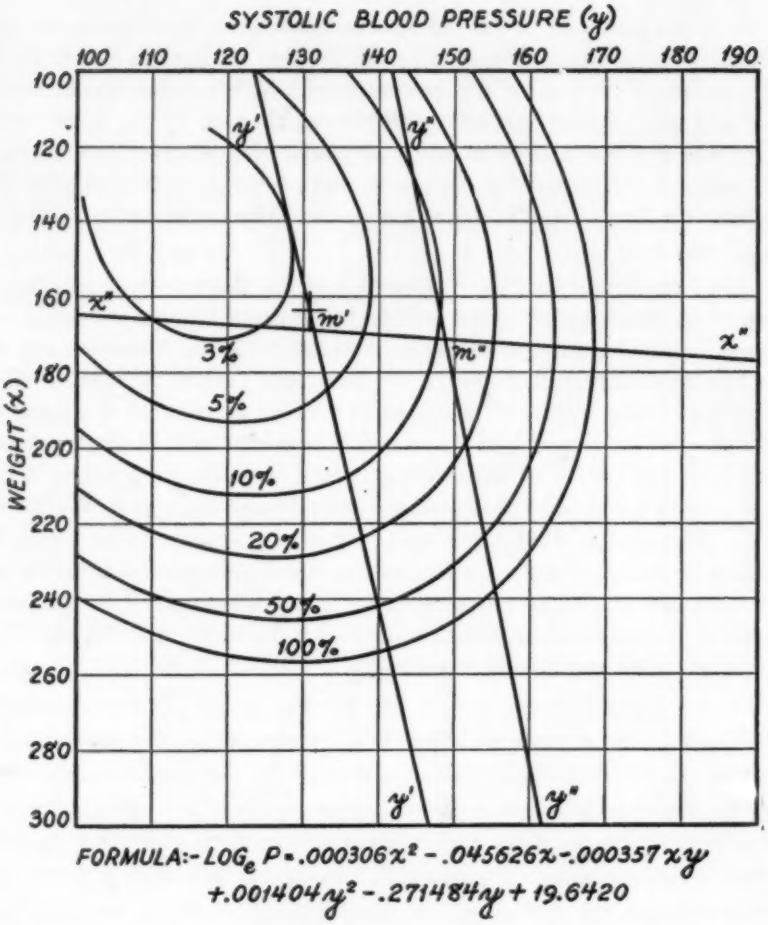

FIG. 8. DISTRIBUTION IN SYSTOLIC-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 46-55 OF DEGENERATIVE CARDIOVASCULAR-RENAL DISEASES DIAGNOSED AT THAT AGE OR LATER IN LIFE. AVERAGE, 8.99%.

The y' and y'' lines are systolic on weight regressions of the general and disease groups; x'' the weight on systolic regression of the latter. m' and m'' are the mean points of systolic and weight.

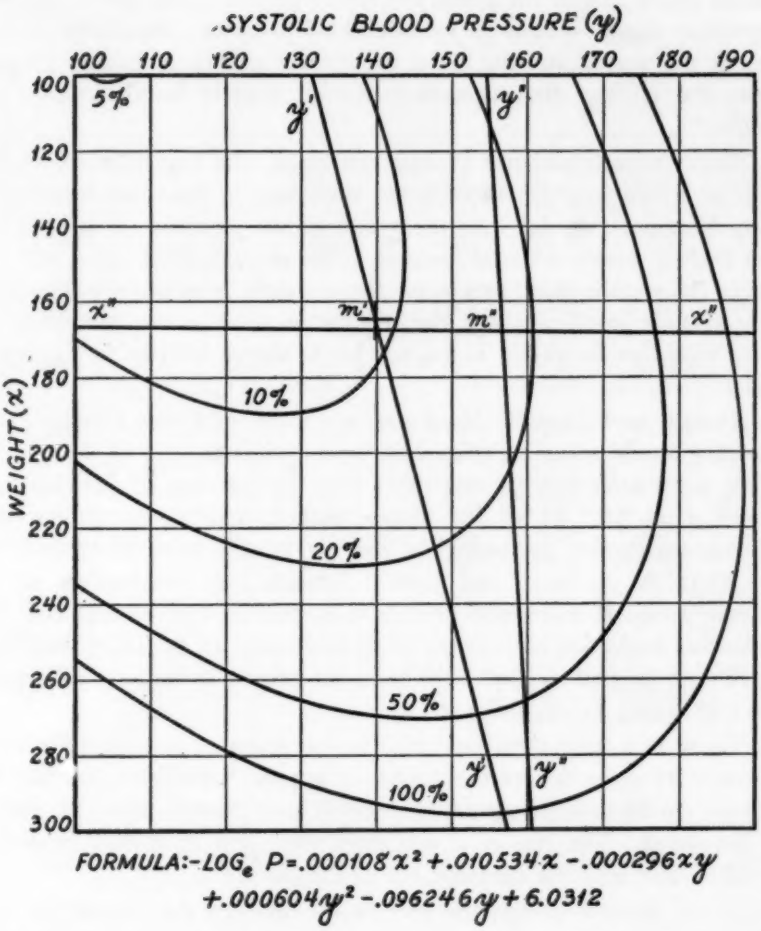

FIG. 9. DISTRIBUTION IN SYSTOLIC-WEIGHT ZONES OF THE GENERAL GROUP AT AGE 56-64 OF DEGENERATIVE CARDIOVASCULAR-RENAL DISEASES DIAGNOSED AT THAT AGE OR LATER IN LIFE. AVERAGE, 16.02%.

The y' and y'' lines are systolic on weight regressions of the general and disease groups; x'' the weight on systolic regression of the latter. m' and m'' are the mean points of systolic and weight.

To read the percentage distribution of the systolic blood pressure, use the upper part of the graph preferably passing along the x'' line, the regression one of weight on systolic blood pressure. Similarly to read that of the weight, use the lower part of the graph, preferably passing along the y'' line, the regression one of systolic blood pressure on weight.*

Since these experience groups are small, the probable errors are large and consequently the expected variations in the exact location of these lines are material. As the graph stands, however, it is apparent that there is a very material increase in the percentage of cases with the rise in the systolic blood pressure. Since weight is so slightly correlated with systolic blood pressure, the relative increase in the percentage of cases with that in weight is not so clearly shown here as in Figures 6 and 7.

Systolic and diastolic blood pressure.—As with the systolic blood pressure, there is but little increase in the diastolic one of the general group until after age 45, and more than 50 per cent of the total rise occurs after 55. If all the known pathological cardiovascular-renal cases are excluded, the means by the age groups from 26-35 to 56-64 are 80.33, 80.52, 82.21, and 84.60. At each age, the diastolic of the disease group is materially greater than that of the general one, the difference beginning with 26-35 being 5.38 mm., 11.41, 10.25, and 8.05. Excluding the pathological cases from the general group, the differences are 5.47, 11.83, 11.21, and 9.36.

There is a very significant rise in the mean of the diastolic blood pressure 10 years before the disease is apparent clinically, but like the systolic the diastolic continues to increase until the age when the disease is diagnosed. The rise in it was then coincident or closely related in point of time with the development of the disease.

In the general group, the correlation between the systolic and diastolic increased from a rather low positive coefficient of .2590 at 21-25, to a high one of .7366 at 56-64, showing a closer association among the older men, possibly the result of degenerative changes. The correlation is higher among the diseased group than the general one in practically every age, indicating again a pathological association.

* The percentage distributions of the upper and lower segments are computed by the use of the plus and minus roots of the radical, or by solving the quadratic for either variable in terms of the other one.

TABLE 9

Constants for Systolic and Diastolic Blood Pressure of the General Group of Army Officers and Also of Those Who had a Degenerative Cardio-vascular-Renal Disease Either at the Age When Measured or Later in Life.

DATA AT AGE	26-35		36-45			
				Disease		
	General Group	Disease at age or 10 yrs. later	General Group	At age	10 yrs. later	At age or 10 yrs. later
Mean:						
Systolic	123.78 ± .16	133.56 ± 2.57	125.91 ± .15	141.53 ± 2.70	139.60 ± 1.72	140.43 ± 1.52
Diastolic	80.42 ± .14	85.80 ± 1.89	80.94 ± .12	93.14 ± 1.72	91.75 ± 1.28	92.35 ± 1.04
Stand. Dev.:						
Systolic	9.28 ± .11	19.07 ± 1.82	11.00 ± .11	24.02 ± 1.91	17.69 ± 1.22	20.66 ± 1.08
Diastolic	8.30 ± .10	14.01 ± 1.34	8.55 ± .08	15.30 ± 1.22	13.13 ± .90	14.12 ± .73
Coeff. of Var.:						
Systolic	7.50 ± .093	14.28 ± 1.39	8.74 ± .086	16.97 ± 1.39	12.67 ± .89	14.71 ± .78
Diastolic	10.32 ± .13	16.33 ± 1.60	10.56 ± .10	16.43 ± 1.34	14.31 ± 1.01	15.29 ± .81
Correlation	.4828 ±.0134	.8141 ±.0455	.6141 ±.0087	.8018 ±.0403	.5598 ±.0669	.6933 ±.0382
Number	1,508	25	2,366	36	48	84

DATA AT AGE	46-55				56-64	
		Disease				
	General Group	At age	10 yrs. later	At age or 10 yrs. later	General Group	Disease at age or 10 yrs. later
Mean:						
Systolic	130.90 ± .23	150.90 ± 1.43	144.33 ± 2.05	148.58 ± 1.19	139.77 ± .57	155.93 ± 2.15
Diastolic	83.17 ± .16	94.94 ± .82	90.65 ± 1.13	93.42 ± .67	85.91 ± .34	93.96 ± 1.17
Stand. Dev.:						
Systolic	14.32 ± .18	20.39 ± 1.01	21.71 ± 1.45	21.10 ± .84	19.70 ± .41	27.29 ± 1.52
Diastolic	9.57 ± .12	11.73 ± .58	11.97 ± .80	11.99 ± .48	11.63 ± .24	14.83 ± .83
Coeff. of Var.:						
Systolic	10.94 ± .14	13.51 ± .68	15.04 ± 1.03	14.20 ± .58	14.09 ± .30	17.50 ± 1.01
Diastolic	11.51 ± .15	12.36 ± .62	13.20 ± .90	12.83 ± .52	13.54 ± .29	15.78 ± .90
Correlation	.7264 ±.0078	.6376 ±.0415	.7528 ±.0409	.6884 ±.0296	.7366 ±.0136	.7109 ±.0391
Number	1,677	93	51	144	522	73

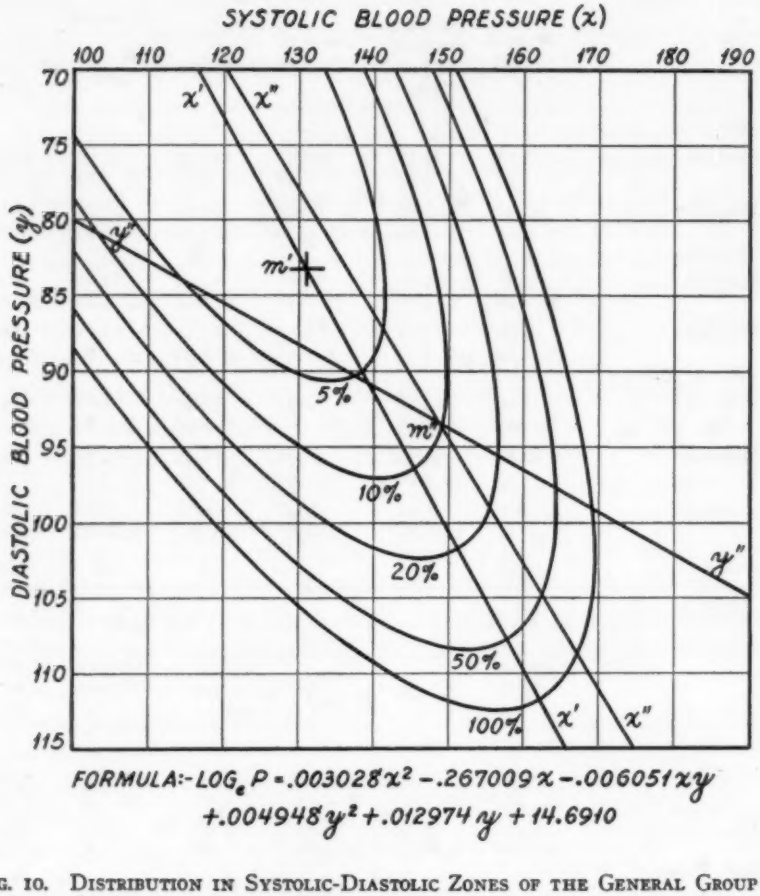

FIG. 10. DISTRIBUTION IN SYSTOLIC-DIASTOLIC ZONES OF THE GENERAL GROUP AT AGE 46-55 OF DEGENERATIVE CARDIOVASCULAR-RENAL DISEASES DIAGNOSED AT THAT AGE OR LATER IN LIFE. AVERAGE, 8.59%.

x' and x'' are systolic on diastolic regression lines of the general and disease groups; y'' is the diastolic on systolic regression line of the latter. m' and m'' are the mean points of systolic-diastolic.

Figure 10 shows in the systolic-diastolic zones of the general group at age 46-55 the percentage distribution of the diseased cases, diagnosed either at that age or later. The average of the diseased in the general group was 8.59 per cent. To read the systolic distribution use the right and upper segment preferably passing along y'' line, the regression one of the diastolic on systolic. Similarly to read the diastolic, use the left and lower segment preferably passing along the x'' line, the regression one of the systolic on diastolic. This graph shows the rapid increase in the percentage of the diseased cases as either the systolic or diastolic increases. Since these experience groups are small and the probable errors are large, the expected variations in the exact location of the percentage lines are material.

CANCER AND OTHER MALIGNANT TUMORS

During this experience, cancer and other malignant tumors caused 84 deaths among the 5,000 officers. The death rate per 1,000 years of exposure was 0.84 as compared with 1.08 among white males of the same age in the registration area during the years 1919-1921. They stood second to nephritis, which caused 128 deaths. The death rates by age groups were: 21-30, .05; 31-40, .09; 41-50, .56; 51-60, 1.45; 61-70, 4.18; and 71-90, 7.10. Only one officer was retired for this disease.

The height-weight experience here is limited to 68 cases. Consequently the findings cannot be considered as very significant, and certainly not as conclusive. An examination of the data in Table 10 shows that in every age group with the exception of one, namely, 56-64, the stature of cancer cases is less than that of the general group. While the differences as measured by their probable errors are apparently not significant, the persistence of them would seem to imply real ones. So far as can be determined from these data, there was no significant or uniform difference between mean weights of the cancer cases and those of the general group, when the disease was diagnosed either at that age or later in life.

NERVOUS AND MENTAL DISEASES

Nervous and mental diseases were the most frequent cause of permanent disability during this experience. They caused a total of 215 retirements for disability as compared with 140 by diseases of the circulatory system. If, however, the nephritis cases are added to the circulatory, the total for the group is 218. Of the 215 cases retired on account of nervous and mental diseases, 133 were for neurasthenia,

TABLE 10

*Constants for Stature and Weight of Army Officers Who had a Cancer or Other Malignant Tumor Either at the Age When Weighed and Measured or Later in Life.**

DATA AT AGE	21-25	26-35	36-45	46-55		56-64
Disease:	At a later age	At age or later	At age or later	At age	At age or later	At age or later
Mean:						
Stature	68.09 ± .22	68.68 ± .24	68.68 ± .23	68.75 ± .25	68.66 ± .20	68.66 ± .29
Weight	144.32 ± 2.66	155.84 ± 2.49	157.05 ± 2.74	158.07 ± 2.88	158.89 ± 2.01	161.59 ± 2.60
Weight at 70 inches	156.09 ± 2.30	164.56 ± 1.95	165.93 ± 2.32	164.98 ± 2.52	164.73 ± 1.82	162.45 ± 2.59
Stand. Dev.:						
Stature	1.50 ± .15	1.94 ± .17	1.99 ± .16	1.98 ± .18	2.02 ± .14	2.35 ± .21
Weight	18.49 ± 1.88	20.51 ± 1.76	24.74 ± 1.64	22.59 ± 2.04	21.74 ± 1.29	20.73 ± 1.83
Coeff. of Var.:						
Stature	2.21 ± .22	2.83 ± .24	2.89 ± .23	2.87 ± .26	2.94 ± .19	3.43 ± .30
Weight	12.81 ± 1.32	13.16 ± 1.15	15.75 ± 1.27	14.29 ± 1.31	13.68 ± .91	12.83 ± 1.16
Correlation	.5004 ±.1078	.6217 ±.0743	.5363 ±.0790	.4849 ±.0975	.4241 ±.0760	.0748 ±.1245
Number	22	31	37	28	53	29

* See Table 2 for the comparative data of the general group.

psychasthenia, and psychoneurosis; 46 for psychosis, and the remaining for scattered causes. The experience shows that officers who were retired as a result of such conditions had at every age an expectancy of life as great as that of the general group. Among the total officers in the Army, during the decade from 1921-30, cardiovascular-renal and mental and nervous diseases each caused the retirement of 125.

In Table 11, we have the results of observations on height and weight of 491 individuals. An examination of these data shows that there was no significant and uniform difference between the mean stature of the mental and nervous group and that of the general one. There was, however, a persistent, uniform, and apparently a real decrease at every age in the mean weight of the men who developed such a disease either then or later.

TABLE 11

*Constants for Stature and Weight of Army Officers Who had a Nervous and Mental Disease Either at the Age When Weighed and Measured or Later in Life.**

DATA AT AGE	21-25		26-35		36-45		46-55		56-64
Disease:	At age	At age or later	At age	At age or later	At age	At age or later	At age	At age or later	At age
Mean:									
Stature	68.63 ± .27	68.85 ± .10	69.17 ± .12	69.09 ± .08	68.84 ± .10	69.04 ± .09	69.12 ± .16	69.18 ± .14	69.05 ± .25
Weight	145.16 ± 2.53	144.13 ± .69	151.94 ± 1.01	151.22 ± .72	155.34 ± 1.02	155.96 ± .88	162.02 ± 1.59	161.93 ± 1.43	163.18 ± 2.66
Weight at 70 inches	154.80 ± 1.69	149.05 ± .56	156.07 ± .82	155.75 ± .60	160.75 ± .90	160.31 ± .78	168.22 ± 1.32	166.23 ± 1.23	164.19 ± 2.65
Stand. Dev.:									
Stature	2.17 ± .19	2.16 ± .07	2.16 ± .08	2.17 ± .06	2.14 ± .07	2.21 ± .06	2.26 ± .11	2.25 ± .10	1.77 ± .18
Weight	20.56 ± 1.79	15.72 ± .49	18.55 ± .71	19.37 ± .51	21.02 ± .72	21.55 ± .62	22.59 ± 1.12	22.95 ± 1.01	18.53 ± 1.88
Coeff. of Var.:									
Stature	3.16 ± .28	3.13 ± .10	3.12 ± .12	3.14 ± .08	3.12 ± .11	3.20 ± .09	3.28 ± .16	3.25 ± .14	2.56 ± .26
Weight	14.16 ± 1.26	10.91 ± .34	12.21 ± .47	12.81 ± .34	13.53 ± .47	13.82 ± .41	13.94 ± .71	14.17 ± .64	11.36 ± 1.17
Correlation	.7432 ±.0551	.5876 ±.0289	.5822 ±.0358	.5548 ±.0257	.4772 ±.0376	.4641 ±.0320	.5547 ±.0487	.5139 ±.0459	.1009 ±.1423
Number	30	234	155	329	192	274	92	117	22

* See Table 2 for the comparative data of the general group.

CONCLUSIONS

1. The cases of pulmonary tuberculosis in young men are much more frequent among the thin ones, and especially the tall thin ones.

2. Only about 1 per cent of the officers developed an active pulmonary tuberculosis after forty-six, but the majority of the ones who did were tall and spare.

3. Tuberculous individuals have the characteristic build as much as 20 years before the clinical evidence of the disease appears.

4. Valvular diseases of the heart, which result from acute rheumatic fever and other infectious diseases early in life, are also more frequent among the tall thin individuals at advanced ages as well as in early adult life. The deficiency of weight in such cases is, however, not so pronounced as in the tuberculous ones.

5. Cardiovascular-renal diseases of the degenerative type which develop in later life are the most frequent causes of disability and death. Of the officers who were active at forty-six, 11.4 per cent subsequently had such a disease.

6. The ones who are over-weight after 30 had a much greater disease expectancy than the ones of average or less than average weight.

7. The increase in weight is apparent from 10 to 20 years before the disease has fully developed and there is no additional increase after it appears.

8. It is the excess in weight above the average for age 30 that is harmful.

9. Since both underweight and overweight are undesirable, one's ability to retain the age 30 average, increases his chances of a maximum of health and longevity.

10. Cardiovascular-renal diseases are associated with an increase in the systolic and diastolic blood pressure. The disease expectancy increases rapidly as the blood pressures become greater.

11. Some of the increase in the blood pressure is apparent 10 years before the disease is diagnosed, but it continues to increase with the development of the disease.

12. Although there is a low order positive correlation between weight and systolic blood pressure in the general group, there is little, if any, in cases of cardiovascular-renal diseases among the older men. This correlation is not increased by holding height constant. Possibly

this is due to the very wide scatter of both the systolic blood pressure and weight among men who have one of these diseases.

13. In this experience there was no consistent difference between the mean weight of the officers who had a cancer or other malignant tumor and that of the general group. The mean stature of the cancer group was, however, persistently lower than the one of the general group.

14. The officers who had a mental or nervous disease had at every age a mean weight which was persistently less than that of the general group.

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VITAL INDICES AND THEIR RELATION TO PSYCHOLOGICAL AND SOCIAL FACTORS*

A STUDY OF THE 310 HEALTH AREAS OF NEW YORK CITY WITH REFERENCE TO BIRTH RATE, DEATH RATE, JUVENILE DELINQUENCY, SCHOOL PROGRESS AND INTELLIGENCE.

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The data of social psychology and social statistics are often based upon records of groups rather than upon measures of a single individual. The group index constitutes the unit of study. Vital statistics, birth and death rates, incidence of diseases and defects, or rate of delinquency are stated in terms of indices, percents or permilles of the population. Different communities can be compared with one another on the basis of such rates and indices.

Psychometric and educational measures are usually given in terms of individual scores. In order to compare groups on these variables the scores must be converted into group measures, modes, medians or means, expressing the central tendency of each group.[1]

Group measures may be used not only for the purpose of determining trends over a period of years or for comparing different groups and communities, but also for the purpose of determining correlations between variables. For example, the correlation between the rate of juvenile delinquency and the rate of infant mortality may be determined by correlating two sets of such indices obtained for a number of communities.

One of the difficulties encountered in determining the correlations between indices from a series of communities is the gross difference

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[1] The author wishes to acknowledge the kind coöperation of those who made the study possible: Dr. Otis W. Caldwell, Director, Institute of School Experimentation; Dr. Harold Hotelling, Columbia University; Dr. Eugene A. Nifenecker, Director of Research, New York Board of Education; Mr. Louis Weiner, Statistician, New York Department of Health; Dr. Neva R. Deordorff, Director of Research, New York Welfare Council.

between communities in the conditions under which the indices are obtained. For example, the rates of juvenile delinquency in different communities are not altogether comparable because of differences in laws and court practices that govern juvenile delinquency. For that reason the correlations obtained from sections of one large city are more meaningful. New York City presents ideal conditions for such a study. Although governed by uniform rules and regulations, the city is divided into numerous divisions and subdivisions. The total city is divided into 310 sections known as health areas, with a population of about 23,000 per area. The present study is based on indices and averages obtained from those 310 areas.[2] (Some of the areas were combined and the actual number was therefore reduced to 275.)

The purpose of this investigation was to study a variety of group measures that were obtained for the 310 health areas into which New York City is divided. These group measures consisted of vital indices including birth rate, death rate and infant mortality, psychometric measures including averages of mental ability and scholastic progress; and social factors including the rate of juvenile delinquency and the national composition of the population residing in the area.

THE NATURE OF THE DATA AND THEIR SOURCES

a. The vital statistics data were secured from the Department of Health of New York City for each of the health areas. The figures for the various health areas were converted into indices, Birth rates (per 1,000 of population), Death Rate (per 1,000) and Infant Mortality Rate (per 1,000 live births). These data were for the year 1930.

b. The data concerning the number of native and foreign born, and the number of white and colored were secured from the Federal Census of 1930. Again, the numbers were converted into percentages showing the proportion of foreign born and the percentage of negroes in each area.

c. The data concerning the proportion of various nationalities in each area were obtained from representative agencies of those groups and from estimates by school principals.

[2] The tabulation of some of the census data by health areas will be found in a publication of the New York Welfare Council. The data for the health areas are based on census figures for smaller units, Sanitary Districts, which were set up under the direction of Dr. Walter Laidlaw. A detailed description of the health areas will be found in the *American Journal of Public Health,* XX, 4, April, 1930, by G. J. Drolet and Dr. William H. Guilfoy in an article entitled *"Organization of Local Health Area Statistics in New York City."*

d. The data concerning juvenile delinquency were secured from Dr. H. L. Shulman of the Crime Commission, who collected the data for the New York Welfare Council. These figures were available for each health area in Manhattan and Bronx. The data were not comparable because of differences in the size of the population. We therefore computed for each health area an Index of Delinquency based on the number of delinquents coming from each health area in 1930, divided by the number of children of court age (6-15) in that area. The latter figures were secured from the Federal Census data.

e. The data concerning school progress, attendance, and average intelligence were obtained from the Department of Research of the Board of Education of New York City:

1. The measure of scholastic progress in each school was expressed in terms of the ratio of accelerated to retarded pupils. These data included the total school child population of the city. The measure of school attendance was based on the daily percentage attendance in each school.[3]

2. Among the most significant variables included in this study was the measure of mental ability. This was the result of an extensive city-wide testing program. The pupils of grade VA and VB of all schools of the city were examined with the National Intelligence Test and the Pintner Rapid Survey Test. The number of pupils tested was 100,153. For each school the average mental age and intelligence quotient were based on the composite result of the two intelligence tests.

The measures of school progress, attendance and average intelligence were computed for each area from the records of the school or schools located in the area.

The vital indices were based on the total population of each area and thus include the records of 6,930,446 individuals, the total population of New York City, as of 1930. The same holds true for the data concerning national composition of the areas. The figures of school progress and attendance include the total child population of each area enrolled in the public schools of the city.

The measure of mental ability is based on the records of the total number of pupils of each area, enrolled in grades VA and VB of the

[3] This was based on the daily attendance during the month of November which was found to be more reliable than the daily attendance based on the whole year.

public schools. Grade V is considered the most representative of the grades and includes a larger percentage of the total number of pupils than any other grade. The average of mental ability based on the records of this grade may therefore be taken as a fairly representative sample of the ability of the total school population of a given area.

In order to reduce the complications resulting from race differences, all areas with a predominantly negro population were either treated separately or excluded altogether.

RESULTS

The results will be presented in three sections, corresponding to the three major considerations of this study:

A. The distribution of the variables, the central tendency and variability of each of the measures.

B. The interrelationship of the variables and the possibility of predicting one from another.

C. An analysis of the intercorrelations with the view of determining the existence of general or group factors and the contribution of the common factor to each variable.

A. Distribution of the Indices

The first question to be considered was the distribution of the indices obtained from the 275 areas of the city for each of the variables. If the respective areas were random samples of the total city population, the index of each area would have approximated the index of the total city, with minor fluctuations due to errors of sampling. The actual distributions revealed marked variations among the areas in each of the variables.

The distribution of the vital indices in 275 areas is shown in Table 1. In view of the marked race differences in vital indices the areas with a predominantly Negro population are not included in these and subsequent distributions.

Table 1 indicates that for each one of the vital indices there was considerable variation among the areas. Thus the birth rate ranged from 4 per 1000 in some areas to 33 per 1000 in other areas, the death rate ranged from 4 to 23 per 1000 and the rate of infant mortality ranged from 20 to 115 per 1000 live births.

The variations indicate that as far as vital indices are concerned the individual areas are certainly not random samples of the city as a whole, but have definite characteristics of their own. The indices of

TABLE 1

Distribution of Vital Indices in 275 Health Areas in New York City

BIRTH RATE PER 1,000 POPULATION		DEATH RATE PER 1,000 POPULATION		INFANT MORTALITY PER 1,000 LIVE BIRTHS	
Scale	Frequency	Scale	Frequency	Scale	Frequency
33.0	2	23	1	115	4
31.5	1	22	1	110	0
30.0	0	21	0	105	1
28.5	0	20	1	100	1
27.0	3	19	2	95	5
25.5	9	18	2	90	8
24.0	7	17	4	85	5
22.5	13	16	4	80	8
21.0	29	15	7	75	11
19.5	29	14	14	70	20
18.0	42	13	23	65	16
16.5	31	12	21	60	17
15.0	30	11	26	55	27
13.5	36	10	29	50	20
12.0	26	9	26	45	39
10.5	5	8	51	40	27
9.0	7	7	38	35	34
7.5	3	6	17	30	16
6.0	1	5	7	25	9
4.5	1	4	1	20	7
Total	275	Total	275	Total	275
Mean	17.87	Mean	10.49	Mean	55.46
S. D.	4.47	S. D.	3.20	S. D.	19.70

the respective areas are probably more significant than the indices for the total city. For example, improvement in the vital index of the total city is not indicative of general improvement. The change may be due to improvement in a few privileged areas, while the conditions in other areas, perhaps in those most in need of attention, may actually become worse.

It is interesting to note that in each case the average of the indices of the various areas corresponds closely to the index of the total city. Thus, the report of the Department of Health for 1930 indicates that the birth rate for the total population of New York was 17.5 per 1000,

the death rate was *10.4* per 1000, and the rate of infant mortality was *56.0* per 1000 live births.

Table 1 gives also the standard deviation of the distribution of indices. By utilizing these standard deviations we can determine whether the difference between two areas is reliable or not.

Table 2 presents the distribution of the averages of mental ability, school progress and percentage of school attendance.

TABLE 2

Distribution of Psychological and Educational Measures in 273 Health Areas in New York City

INTELLIGENCE QUOTIENTS		SCHOOL PROGRESS		PERCENTAGE ATTENDANCE	
Scale	Frequency	Scale	Frequency	Scale	Frequency
118	2	210	0	99.0	0
114	11	190	1	98.2	0
110	18	170	2	97.4	3
106	42	150	5	96.6	33
102	46	130	4	95.8	89
98	57	110	9	95.0	88
94	40	90	23	94.2	37
90	29	70	29	93.4	14
86	14	50	44	92.6	5
82	7	30	84	91.8	2
78	6	10	67	91.0	0
74	1	below 10	5	below 91.0	2
Total	273	Total	273	Total	273
Mean	100.42	Mean	54.50	Mean	95.60
S. D.	8.28	S. D.	35.53	S. D.	1.04

Psychological measures: Table 2 reveals that the distribution of average intelligence quotients, in the 273 health areas was fairly normal. It ranged from less than 76 to 120, with an average of 100 and a standard deviation of 8.

It appears that there are areas in the city in which the *average* intelligence is at the level usually referred to as "borderline" while in other areas the *average* intelligence reaches the level usually considered as "very superior."

The distribution of the 273 averages is quite similar to the distribution of the individual scores. The mean of the scores of 100,153

children to whom the intelligence tests were given was 100.2 and the standard deviation of these scores was 20.9.

Homogeneity with regard to intelligence: If the areas were random samples of the total population then the S. D. of the means would have equalled approximately the S. D. dis./$\sqrt{n}$, where S. D. dis. is the S. D. of the total distribution and n is the total population divided by the number of areas. In our case the S. D. of the means would have been 1.09. Actually, the S. D. of the means was 8.28 or more than 7 times as high as the expected. This indicates that the homogeneity with regard to intelligence of the people residing in the same area is on the average, very much higher than that to be found among a similar number of people selected at random. The correlation due to homogeneity, the ratio of the two sigmas, was .40 ± .03.

Educational measures: The distribution of the rates of school progress does not have the same characteristics as the distribution of intelligence scores. The rate of school progress was measured by the ratio of the number of accelerated pupils to the number of retarded pupils within each area.

Acceleration and retardation are not determined on the basis of objective measures and often depend upon local conditions and upon the educational views of the teachers, principal and superintendent. Promotion is often determined by relative rather than actual achievement. Thus a pupil of "average" achievement placed in a school of dull pupils may soon become accelerated. The same pupil placed in a school of relatively bright pupils will tend to become retarded. For these reasons the distribution is not "normal." It is definitely skewed to the right, with marked clustering at the lower (retardation) end of the scale.

The average of the 273 areas in rate of progress was 54.5, that is, the average area has about 55 accelerated to every 100 retarded pupils. There was, however, tremendous variability among the health areas in the rate of progress. The range was from nearly zero (where there were no accelerated pupils at all) to nearly 200 (where the number of accelerated pupils was about twice the number of retarded pupils). The standard deviation of these rates of progress was 35.5. The school status of the total number of pupils in the average area was as follows:

Percentage retarded	28.8
Percentage normal	55.9
Percentage accelerated	15.3

These proportions for the total city, according to the report of the Board of Education, based on the same data as used in this study, were:

Percentage retarded	28.9
Percentage normal	52.8
Percentage accelerated	13.9
No data	4.9

The distribution of the rates of attendance was also somewhat asymmetrical.

The percentage of daily attendance (November, 1931) in the average area was 95.6. The range of the percentages of attendance was from less than 90 per cent to more than 98 per cent. The S. D. of the rates of attendance in 273 areas was 1.04.

The distributions of indices of social adjustment: Table 3 presents the distribution of the rate of juvenile delinquency and the percentage of foreign born in the health areas of New York. The *delinquency rate* is the number of juvenile delinquents arraigned in a given area during 1930, per 1000 children of court age (6-15) of that area. This measure was available only for Manhattan and the Bronx. The *proportion foreign born* is the ratio of the number of foreign born white to the total population of each area.

TABLE 3

Distribution of Two Social Indices in the Health Areas of New York City

PERCENTAGE FOREIGN BORN		JUVENILE DELINQUENTS PER 1,000 OF COURT AGE	
Scale	Frequency	Scale	Frequency
54	6	22	1
50	6	20	1
46	21	18	1
42	29	16	4
38	34	14	5
34	33	12	6
30	37	10	10
26	47	8	21
22	30	6	24
18	21	4	23
14	9	2	24
10	2	0	6
Total	275	Total	126
Mean	33.77	Mean	6.54
S. D.	9.57	S. D.	4.40

Rate of juvenile delinquency: It will be seen from Table 3 that the average rate of delinquency was 6.5 per 1000 children of court age, 6-15. This was based on the data for Manhattan and the Bronx including 126 health areas. The range was from zero, for areas in which there was not a single juvenile delinquent, to 22 for areas in which 22 out of every 1000 children of age 6-15 were arraigned before the children's court as alleged delinquents. The standard deviation of the rates for 126 areas was 4.4, indicative of a marked variation among the areas in the rate of juvenile delinquency.

Proportion of foreign born: Table 3 shows that the proportion of foreign born in the average area was about one-third of the area's population, 33.77 per cent. The range was considerable, from 10 per cent to 58 per cent. These figures based on the data of the Federal Census of 1930 reveal that there are no areas in New York City where the foreign born population is as high as 58 per cent. The proportion of natives is at least 42 per cent. Apparently the foreign group residing in New York City tends to disseminate throughout the city, somewhat clustering in certain areas. According to the Census report the number of foreign born in New York City was (in 1930) 2,293,400 out of the total of 6,930,446 or 33.09 per cent. This percentage is approximately the same as the average percentage we found in the study of 275 health areas and indicates the tendency on the part of the foreign born to be somewhat "normally" distributed throughout the city. The standard deviation of the proportion foreign born was 9.57 and the distribution is only slightly asymmetrical.

B. Inter-relationship Among Vital Indices, Psychological and Educational Measures, and Social Factors

The preceding considerations dealt with the distribution of each of the variables in the various health areas of the city. We shall now turn to a consideration of the inter-relationship among these variables. The correlations (Pearson product-moment) between each variable and the others were computed. The coefficients of correlation are shown in Table 4. The correlations are based on the records of 275 areas with the exception of the correlations with juvenile delinquency which are based on 126 areas.* This table presents all the intercorrelations among

* The delinquency rates were subsequently obtained for the other areas. The correlations were practically unchanged.

the eight variables. The upper-right half of the table gives the correlations and the lower-left half of the table gives the probable errors of the respective correlations. The table reads as follows: the correlation between Intelligence and School Progress is .6986 and the probable error of the correlation is .02.

Table 4 reveals some interesting relationships among the variables. Among the significant correlations (those which are at least four times their probable errors) are the following:

1. Intelligence correlates positively with school progress to the extent of .70 and with regularity of school attendance .16. Intelligence correlates negatively with birth rate to the extent of —.34, with death rate —.43, with the rate of infant mortality —.51, and with the rate of juvenile delinquency —.57.

2. School progress correlates positively with intelligence .70 and negatively with birth rate —.30, with death rate —.34, with infant mortality —.46, and with juvenile delinquency —.43. It will be noted that each of these correlations is negative but not as markedly negative as were the correlations of these variables with intelligence. Apparently school progress is less related to the stamina and vitality of a group than is intelligence. This is also seen from the other less significant correlations. Thus while intelligence correlates somewhat positively with the proportion of foreign born (.10) school progress correlates negatively with it (—.16).

It was quite surprising to find that regularity of school attendance correlates more with intelligence (.16) than it correlates with school progress (.09).

3. Regularity of school attendance correlates positively with intelligence .16, and with the proportion of foreign born .17. It correlates negatively with death rate —.25, with infant mortality —.22 and with juvenile delinquency —.27.

4. Birth rate correlates negatively with intelligence —.34, with school progress —.30. It correlates positively with juvenile delinquency to the extent of .28, indicating that the areas with higher birth rate also tend to have a higher rate of juvenile delinquency.

5. Death rate shows correlations similar to those of birth rate. It correlates negatively with intelligence —.43, with school progress —.34, with school attendance —.25. It correlates positively with juvenile delinquency .59 and with infant mortality .58. The latter correlation is

TABLE 4

Intercorrelation Between Vital Indices and Psychological and Social Factors

	1 INTELLIGENCE QUOTIENT	2 SCHOOL PROGRESS	3 SCHOOL ATTENDANCE	4 BIRTH RATE	5 DEATH RATE	6 INFANT MORTALITY	7 JUVENILE DELINQUENCY	8 PROPORTION FOREIGN BORN
1. Intelligence Quotient	..	.6986	.1600	—.3409	—.4266	—.5125	—.5681	.0964
2. School Progress	.02	....	.0874	—.3025	—.3419	—.4575	—.4268	—.1582
3. School Attendance	.04	.04	....	—.0453	—.2543	—.2242	—.2695	.1693
4. Birth Rate	.04	.04	.04	....	.0568	.0397	.2812	.0063
5. Death Rate	.03	.04	.04	.04	....	.5772	.5883	.0147
6. Infant Mortality	.03	.03	.04	.04	.03	....	.5787	.0660
7. Juvenile Delinquency	.04	.05	.06	.05	.04	.04	....	—.1225
8. Proportion Foreign Born..	.04	.04	.04	.04	.04	.04	.06	....

somewhat spurious as the number of dying infants enters also in the total death rate.

6. Infant mortality is similar in its correlations to birth rate and death rate. It correlates negatively with intelligence —.51, with school progress —.46, and with school attendance —.22. It correlates positively with death rate .58 and with juvenile delinquency .58.

7. The rate of juvenile delinquency correlates negatively with intelligence —.57, with school progress —.43, and with school attendance —.27. It correlates positively with birth rate .28, with death rate .59 and with infant mortality .58.

8. The proportion of foreign born does not show any marked correlations with the other variables. It correlates positively with intelligence .10, and with school attendance .17. It correlates negatively with school progress —.16 and with juvenile delinquency —.12. The last correlation, indicating that the areas with high proportions of foreign born tend to have a lower rate of juvenile delinquency, is of particular interest in the light of frequent, though unsubstantiated assertions that criminality is more frequent among the foreign elements of the population.

Analysis of Relationship: The coefficient of correlation indicates the average degree of co-variance between two sets of measures, the extent to which variation in one is accompanied by variation in the other. Like any other average, it reveals only the central tendency but not the total relationship. Without knowing the type of relationship, whether it is linear or curvilinear, the coefficient of correlation has as little meaning as an average based on *scores* whose distribution is unknown. A more comprehensive picture is revealed from the actual line of regression going through the means of the arrays and from the correlation ratio based upon these means. The line of means was obtained for some of the more significant correlations. In most instances the range was divided into nine intervals and the averages of these arrays were computed. In a few instances a smaller number of arrays were used in order to eliminate fluctuations.

Relationship with Intelligence: Figures 1 to 5 show the relationship between different levels of intelligence and other variables including birth rate, death rate, infant mortality, juvenile delinquency and school progress. Figure 1 is to read as follows: Those areas in which the

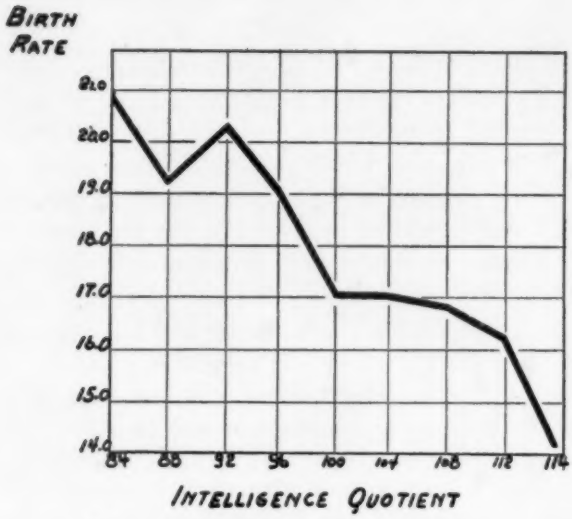

FIG. 1. REGRESSION OF BIRTH RATE ON INTELLIGENCE QUOTIENT

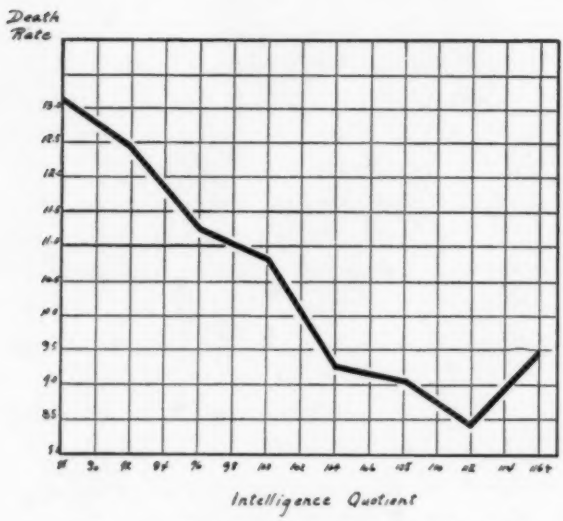

FIG. 2. REGRESSION OF DEATH RATE ON INTELLIGENCE QUOTIENT

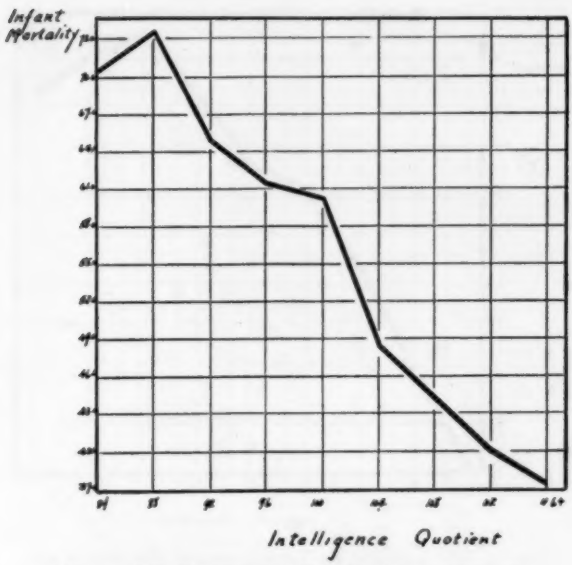

FIG. 3. REGRESSION OF INFANT MORTALITY ON INTELLIGENCE QUOTIENT

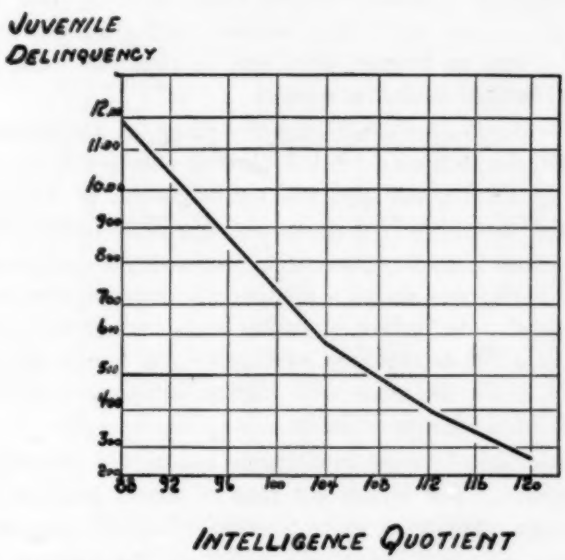

FIG. 4. REGRESSION OF JUVENILE DELINQUENCY ON INTELLIGENCE QUOTIENT

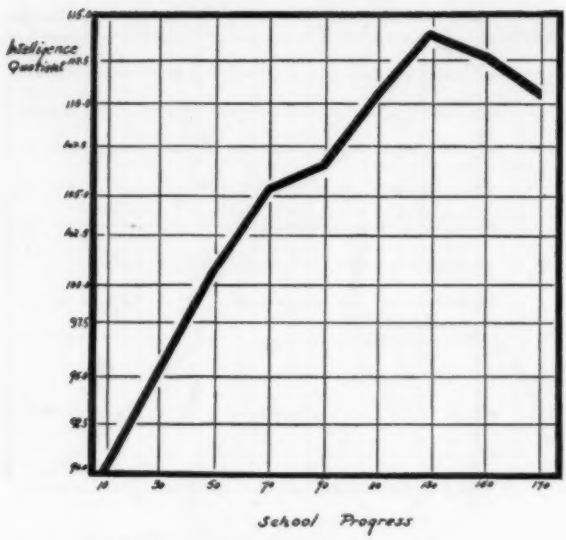

FIG. 5. REGRESSION OF INTELLIGENCE QUOTIENT ON SCHOOL PROGRESS

average intelligence quotients ranged from 84 to 87 had an average birth rate of 20.9 per 1000, those whose intelligence quotients ranged from 88 to 91 had an average birth rate of 19.6, etc. The rest of the figures are to be read in similar manner.

For convenience of comparisons the range of intelligence may be divided into three sections: the dull (lowest obtained I. Q. to 95), the average (I. Q. from 96 to 107) and the bright (I. Q. 108 to highest). The number of areas involved in each array will be found in Table 2.

It will be seen from Figures 1 to 4 that, with the exception of minor fluctuations intelligence shows a consistently negative relationship with birth rate, death rate, infant mortality and juvenile delinquency. In those areas in which the average intelligence was lowest the above rates were highest, while the areas with highest intelligence had the lowest rates in each of the above variables.

The relationship between intelligence and school progress was consistently positive. The higher the rate of school progress the higher was the average intelligence up to the level of school progress indicated by a quotient of 130. From that point on, the average intelligence ceased to increase and shows a slight tendency to decrease. (See Fig. 5.)

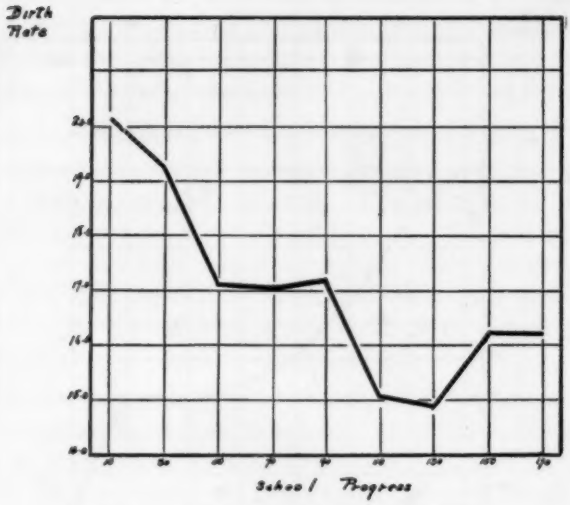

FIG. 6. REGRESSION OF BIRTH RATE ON SCHOOL PROGRESS

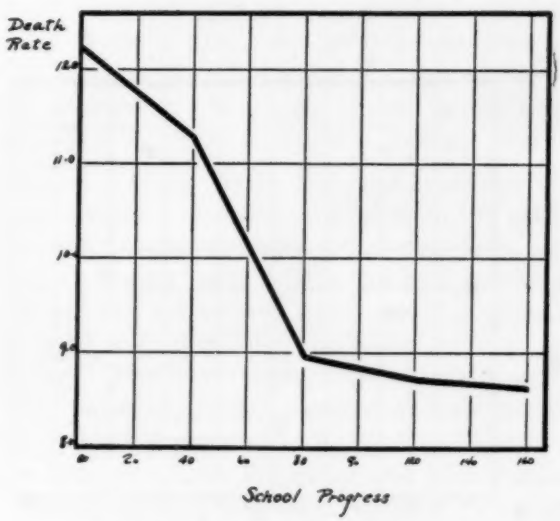

FIG. 7. REGRESSION OF DEATH RATE ON SCHOOL PROGRESS

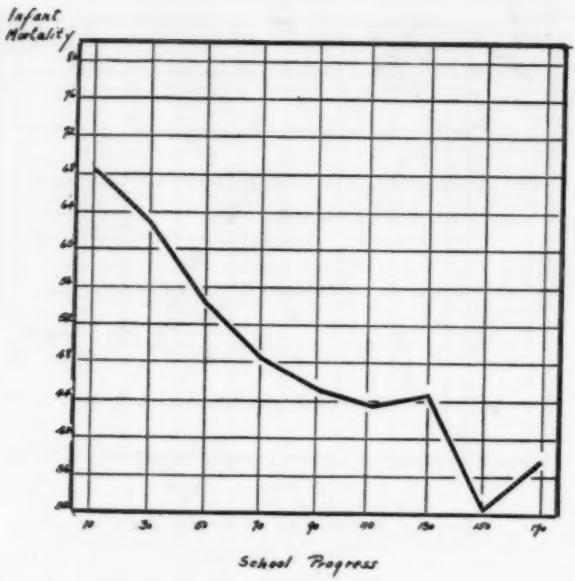

FIG. 8. REGRESSION OF INFANT MORTALITY ON SCHOOL PROGRESS

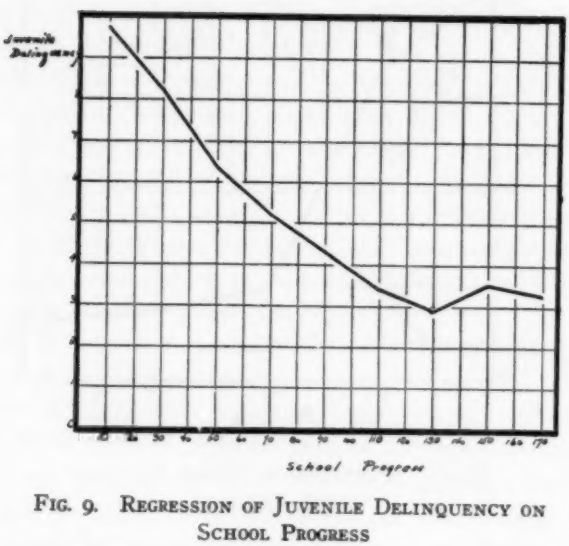

FIG. 9. REGRESSION OF JUVENILE DELINQUENCY ON SCHOOL PROGRESS

School Progress: Next to the records of intelligence the measure of school progress showed the highest correlations with the other variables. Figures 5 to 9 show the relationship between school progress and intelligence, birth rate, death rate, infant mortality and juvenile delinquency.

Figure 5 has been discussed in the preceding paragraphs. Figures 6 to 9 indicate that the relationship between the rate of school progress and the vital indices is definitely negative. The relationship is not as markedly negative as was the case with intelligence. The areas in which the rate of progress was high (more accelerated than retarded pupils) have a lower birth rate, death rate, infant mortality rate, and juvenile delinquency rate than the areas in which the rate of school progress is low.

In order to make these comparisons clearer, the 275 areas of the city were divided into three categories: areas with a rate of school progress above 100, where the number of accelerated pupils exceeds the number of retarded pupils (there were 31 areas in this group), areas with a normal rate of progress, where the rate ranged from 40 to 100 (there were 127 areas in this group), and areas with a very low rate of progress, where the rate ranged from 10 to 40 (116 areas). The averages of each of these groups on each of the variables was determined. These are shown in Table 5. The relationship is consistent without a single exception. The accelerated areas had a higher average of intelligence and school attendance and a lower average of birth rate, death rate, infant mortality, and juvenile delinquency than the retarded areas.

School Attendance: None of the records included in this study showed a marked correlation with school attendance. It is interesting to note that the highest correlation with school attendance was shown by the juvenile delinquency rates. This correlation was negative, —.2695 and indicates that the areas in which school attendance is low, i. e., the percentage of daily absentees is high, tend to have also a high proportion of young delinquents. Similarly, there was a negative correlation of —.2242 between school attendance and the rate of infant mortality. This relationship is shown in Figure 10.

It will be seen from Figure 10 that school attendance decreases with increase in infant mortality up to point 85 in the latter. From that point on school attendance tends to increase. It is likely that school attendance is at its highest in the average population and that among both the lower and the higher levels of society the regularity of school attendance

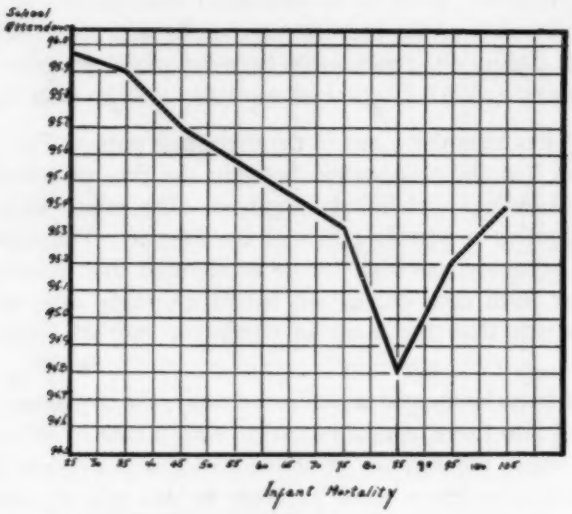

FIG. 10. REGRESSION OF SCHOOL ATTENDANCE ON INFANT MORTALITY

is impaired. In the former group, attendance is lower probably because of the inadequate care and attention and in the latter group because of excessive care and attention on the part of the parents towards their children.

TABLE 5

School Progress and Its Relation to Social, Psychological and Vital Indices

NATURE OF INDEX	RETARDED AREAS	NORMAL AREAS	ACCELERATED AREAS
1. Birth Rate	19.4	17.2	15.5
2. Death Rate	11.7	9.9	8.7
3. Infant Mortality	62.5	48.0	40.6
4. Juvenile Delinquency	7.9	4.7	2.8
5. Intelligence Quotient	94.5	103.2	111.7
6. School Attendance	95.5	95.7	95.7
7. Proportion Foreign Born	35.4	33.1	31.3
Number of Health Areas	116	127	30

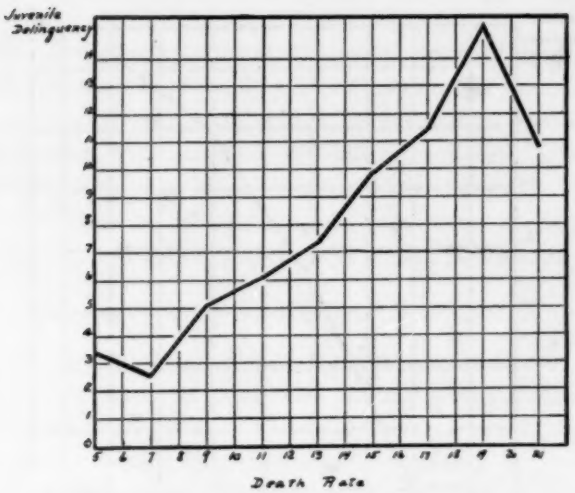

FIG. 11. REGRESSION OF JUVENILE DELINQUENCY ON DEATH RATE

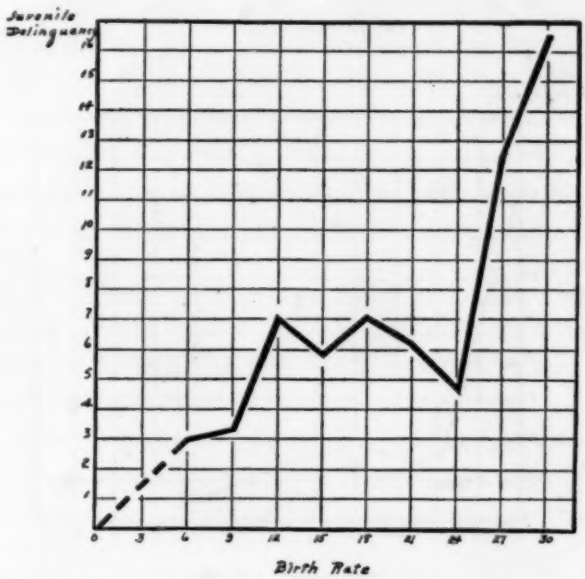

FIG. 12. REGRESSION OF JUVENILE DELINQUENCY ON BIRTH RATE

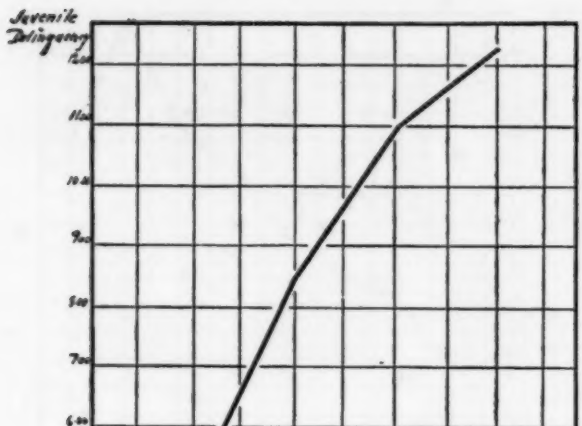

FIG. 13. REGRESSION OF JUVENILE DELINQUENCY ON INFANT MORTALITY

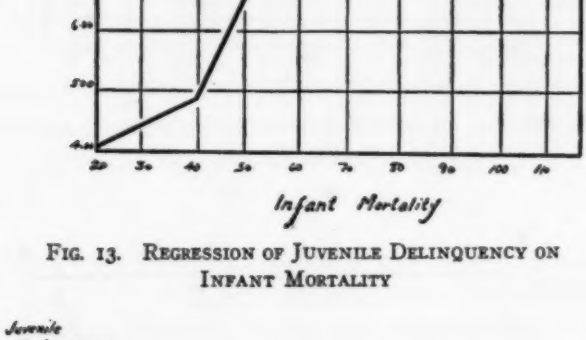

FIG. 14. REGRESSION OF JUVENILE DELINQUENCY ON PERCENTAGE FOREIGN BORN

Juvenile Delinquency: From the social point of view, the rate of juvenile delinquency is probably a better measure of the effectiveness of our educational system, than school progress and school attendance. The relationship between juvenile delinquency and intelligence and school progress was negative as shown in Figures 4 and 9. The relationship between juvenile delinquency and proportion foreign born, birth rate, death rate, and infant mortality is shown in Figures 11 to 14.

C. Analysis of General and Specific Factors

The preceding consideration of the relationship among the variables included in this study revealed one striking result. The "desirable" factors such as intelligence, school progress and school attendance correlate positively among themselves and negatively with the "undesirable" factors. Similarly, the records of death rate, infant mortality and juvenile delinquency correlate positively among themselves and negatively with each of the other group. In other words, all "desirable" factors go together and so do all the "undesirable" ones. If we reverse the indices of the latter group, changing the death rate to an "index of vitality," the rate of infant mortality to an "index of infant survival," and the rate of juvenile delinquency to an "index of juvenile uprightness," (or "juvenile non-delinquency") the correlations will obviously remain unchanged in magnitude, but changed in sign. All the correlations will then be positive.

The problem arises whether the relationship among these variables is due to a series of specific factors, each correlation being the result of some element common to two, and only two, of the variables? or is it due to the existence of some general factor or factors common to the various measures? In other words, are the correlations the results of independent specific bonds or are they, themselves, related to one another due to some general bond?

Professor C. Spearman has proposed a method of analysis to determine whether or not a number of correlations may be viewed as due to a general factor. This method of factor analysis was applied to the data of this study.

Table 6 gives the tetrad difference analysis for six variables. The record of the proportion of foreign born was excluded from the analysis

because it differs qualitatively from the other records. Death rate and infant mortality rate were combined because of the obvious "overlap" between them. The six variables included in the analysis were therefore

1. Birth rate
2. Death rate (including infant mortality)
3. Juvenile delinquency
4. School progress
5. Intelligence quotient
6. Regularity of school attendance.

The 15 inter-correlations and the resulting 45 tetrad differences are given in Table 6. The average correlation was .3403 and the probable error of the tetrad differences, determined by Formula 16A of Spearman's "Abilities of Man," was .0223.

It will be seen from Table 6 that in at least 12 instances the tetrad criterion was not satisfied. This, however, was partly due to "overlap." The correlation between intelligence and school progress was considerably higher than the correlations between any of the other variables. It is obvious that those two measures have much more in common with one another than with the other measures. There is thus present the element of overlap. These two were, therefore, combined. Similarly, birth rate and death rate were combined because they are both vital indices.

As a result of the above combination the table was reduced to four variables:

1. Vital factors (reversed vital indices)
2. Juvenile non-delinquency (reversed juvenile delinquency index)
3. Mental ability (intelligence and school progress)
4. Regularity of school attendance

Table 7 gives the intercorrelations and the tetrad differences for these four variables.

The average correlation was .3895. The median probable error of the tetrad differences was .0248. It will be seen from Table 7 that the tetrad criterion was satisfied and that the largest tetrad difference was only three times the probable error. This indicates that these four variables may be considered as having one factor common to them. This factor may be looked upon as the *developmental status of the group* which expresses itself in the level of physical, mental and social adjustment. The correlation between each of the four variables and the com-

TABLE 6

*Tetrad Difference Analysis of Six Variables**

	1 BIRTH RATE	2 DEATH RATE	3 JUVENILE DELIN-QUENCY	4 SCHOOL PROGRESS	5 INTEL-LIGENCE QUOTIENT	6 SCHOOL ATTEND-ANCE
1. ...	....	.0543	.2812	.3025	.3049	.0453
2. ...	.0543	....	.6571	.4501	.5287	.2694
3. ...	.2812	.6571	....	.4268	.5681	.2695
4. ...	.3025	.4501	.4268	....	.6986	.0874
5. ...	.3049	.5287	.5681	.6986	....	.1600
6. ...	.0453	.2694	.2695	.0874	.1600	....

t_{1234}	—.103393	t_{1345}	—.175598	t_{1456}	.072205
t_{1235}	—.117823	t_{1356}	—.169602	t_{1536}	.051679
t_{1236}	—.061122	t_{1346}	—.015133	t_{1436}	—.045989
t_{1245}	—.121998	t_{1256}	—.099302	t_{1546}	—.022696
t_{1246}	—.076748	t_{1256}	—.015644	t_{1645}	—.061104
t_{1256}	—.073453	t_{1265}	—.015263	t_{1652}	—.058190
t_{1345}	.024596	t_{1254}	.066315	t_{1542}	—.041719
t_{1346}	—.056947	t_{1264}	.005243	t_{1642}	—.062190
t_{1356}	—.037179	t_{1263}	.019254	t_{1653}	—.056436
t_{1456}	.021752	t_{1463}	.016753	t_{1654}	.004999
t_{2345}	.203348	t_{2354}	.233401	t_{2543}	—.030053
t_{2346}	—.063872	t_{2364}	—.057550	t_{2643}	—.006322
t_{2356}	—.037349	t_{2365}	—.047911	t_{2653}	.010562
t_{2456}	.025808	t_{2465}	—.116186	t_{2654}	.141994
t_{3456}	.018636	t_{3465}	—.119985	t_{3654}	.138621

* The variables that yielded negative correlations were reversed, thus changing all negative correlations to positive.

mon factor of developmental status, which will be referred to as D, were as follows:

Correlation between Vital indices and D .7800
Correlation between Mental ability and D .8709
Correlation between Juvenile non-delinquency and D .6640
Correlation between School attendance and D .2351

NATIONAL DIFFERENCES

The present study is based on the total white population of New York City. Areas with a Negro population were treated separately to eliminate the factor of heterogeneity due to possible race differences.

TABLE 7

Tetrad Difference Analysis for Four Composite Variables

	1 VITAL INDICES	2 JUVENILE NON-DELINQUENCY	3 MENTAL ABILITY	4 SCHOOL ATTENDANCE
1. ...	....	.6462	.5927	.1543
2. ...	.6462	....	.5398	.2695
3. ...	.5927	.5398	....	.1342
4. ...	.1543	.2695	.1342	....

t_{1234} ..... —.073013
t_{1342} ..... .003429
t_{1423} ..... —.076442

The white population, however, is far from being homogeneous as it is composed of different national and ethnic groups differing in cultural background as well as in physical and mental characteristics. The correlations presented above are thus to some extent affected by this factor of heterogeneity.

In order to investigate the nature of the national differences among the various health areas of the city, two national groups, the Italians and the Jews, were chosen for study. These two groups are well represented in New York City including more than three million of the total population.* They have distinct characteristics and do not overlap.

The number and percentage of each of these national groups in each of the areas was determined. A special study was then made of those areas in the city where the residents are predominantly of these two national groups. Thirty such areas were found in the city with a population of about three-quarters of a million. In fifteen of these areas 85 to 100 per cent of the population was Italian and in the other fifteen areas 90 to 100 per cent of the population was Jewish.

Table 8 gives the average and variability on the various indices for the two national groups, Italian and Jewish. The table reads as follows:

The average birth rate in the fifteen areas of Italians was 23.15 per 1000 with an S. D. of 2.35, while in the fifteen areas of Jews it was only 18.45 per 1000. The difference was 4.7 and the S. D. of the

* The Jewish population is approximately two million. The Italian population, including those born in Italy and their children, according to the 1930 Census is 1,070,355.

TABLE 8

Indices for Areas of Two National Groups

Factor	ITALIANS		JEWS		Difference	σ Diff.	D/σ Diff.
	Mean	S. D.	Mean	S. D.			
Birth Rate	23.15	2.35	18.45	2.20	+ 4.70	0.835	+ 5.63
Death Rate	10.50	2.92	7.96	1.54	+ 2.54	0.850	+ 3.00
Infant Mortality	58.17	22.28	46.50	12.40	+ 11.67	6.580	+ 1.77
Juvenile Delinquency	8.80	1.89	3.70	1.30	+ 5.10	0.770	+ 6.62
Intelligence Quotient	87.80	5.26	106.20	3.70	— 18.40	1.665	— 11.02
Mental Age	116.70	6.62	135.10	3.82	— 18.40	1.976	— 9.29
School Progress	19.67	10.24	71.00	34.02	— 51.33	9.168	— 5.60
Attendance	95.57	7.79	96.37	6.77	— 0.80	2.665	— 0.30
Foreign Born White	36.33	4.71	45.80	4.25	— 9.47	1.643	— 5.76

difference was 0.835. The critical ratio, the difference in terms of its S. D., was 5.63.

Table 8 reveals that the Italians have a *higher* average than the Jews in birth rate, death rate, infant mortality rate, and rate of juvenile delinquency. All these differences, excepting the difference for infant mortality, were statistically significant. On the other hand, the Italians have a *lower* average than the Jews in intelligence, a lower average mental age, and a lower rate of school progress. All of these differences were statistically significant. The S. D.'s of the Italian areas were generally larger.

Of particular interest was the difference in intelligence, both because of the significance of the results based on objective tests and because of the striking magnitude of the difference. The *highest* average intelligence obtained by any Italian area was 95.0. The *lowest* average intelligence obtained by any Jewish area was 96. The range of the Italian areas was from an average I. Q. of 74 to an average of 95. The range of the Jewish areas was from 96 to 123. There was thus an actual "chasm" between the distribution of the intelligence quotients of the two national groups. The most intelligent of the Italian areas was lower than the least intelligent of the Jewish areas. The average difference was 18 points of I. Q. and more than one and a half year in "mental age." [4]

The difference in school progress was similarly striking. In the Italian areas the ratio of accelerated to retarded pupils was less than 20, that is, there were less than 20 accelerated pupils for every 100 retarded ones. In the Jewish areas there were more than 70 accelerated per every 100 retarded pupils.[5]

It is interesting to note that the Jewish group, which is superior to the Italian on each of the measures, has a higher proportion of foreign born than the latter group. It indicates that, at least as far as these groups are concerned, national differences can hardly be ascribed to differences in the proportion of foreign born.

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[4] The correlation between the proportion of Jews in an area and its average intelligence (including all the 275 areas of the city) was positive .469. The correlation between the proportion of Italians in an area and its average intelligence was negative —.418.

[5] The correlation between the proportion of Jews and school progress was .419. The same correlation with the proportion of Italians was —.483.

SUMMARY

A study was made of the health areas of New York City with reference to birth rate, death rate, infant mortality, juvenile delinquency, intelligence, school progress and attendance, and the proportion of foreign born.

The status of each area with regard to the above measures was determined and the distribution of the areas in each measure was ascertained. The various inter-correlations were computed and analyzed. The following is a summary of the findings revealed by the study:

1. The health areas into which New York City is divided differ enormously from one another in each of the variables studied. The city may well be considered as composed of 310 small communities with a population of some 23,000 in each.

2. The factors included in the study show a marked degree of intercorrelation. Desirable characteristics seem to go together. Areas that excel in "vitality" tend also to excel in intelligence and scholastic attainment and social adjustment.

3. There seems to be evidence of a general factor common to the various factors, measures and indices. This factor represents the developmental status of respective groups residing in the various areas, and is referred to as factor D. The measures that correlate highest with this common factor are those of social adjustment (based on rate of juvenile delinquency), mental ability (based on psychological tests and school progress), and the measures of vital statistics. The measure of regularity of school attendance shows the lowest correlation with the common factor.

4. There seem to be marked national differences in each of the measures. An analysis of the difference between two national groups, the Italians and the Jews, revealed that the Jews were strikingly superior to the Italians on each of the measures and indices.

5. The study indicates that the respective areas of the city have population units quite homogeneous in physical, mental, and social characteristics.

A single index for the city, whether it be in vital statistics or in psychological measures has very little meaning. Statements based on the *average* of vital indices, school progress, and achievement and the like without consideration of the tremendous differences among the different areas and schools are often misleading.

THE PHYSIQUE OF WOMEN STUDENTS AT NEWCOMB COLLEGE OF TULANE UNIVERSITY

IV. STATURE, WEIGHT AND BODY BUILD BY NATIONAL EXTRACTION

BY HARLEY N. GOULD

Department of Biology, Newcomb College of Tulane University

Anthropometric studies of various groups of college women in the United States have shown significant differences in stature, weight and body build in different parts of the country. Geography, climate and other environmental influences have been indicated as responsible in part for such differences. The suggestion presents itself that if the Newcomb College students, whose physical measurements have already been analyzed as a whole, by ages, and by year of entrance to college (Gould, '30a; Gould and Lockhart, '30b; and additional in press) could now be differentiated according to the national origin of their ancestors, the study of the groups might be of some value; for this college, located in a subtropical region and drawing its attendants from the near vicinity, contains students of lower stature, lighter weight and more slender build than the women of northern colleges. Such a study might indicate to what extent the stature and body build of the young women from the Gulf Coast states were affected by racial peculiarities, rather than by geographical influences.

Each student, on being examined by the physical education department, is required to give the principal national origins of both parents. Consequently we have, for each subject, her own estimate on this point. No claim can be made for real accuracy of these statements, but they give an opportunity for a rough classification of the elements making up the college population in this Gulf Coast region.

In assembling the data for this study, I have been assisted by Miss Frances Johnson, to whom grateful acknowledgments are due. The transcriptions from the original records have been checked by both of us.

A brief review of the movements of white immigration to the Gulf Coast states will give an idea of the racial stocks from which our material is drawn. French colonization began about 1697, but the actual

influx of French settlers dates from about 1718, with the formation of John Law's "Company of the West." Many of the earliest French immigrants were of poor quality, ill adapted to colonial life, and left few descendants (at least few of pure white stock). Some of the more successful were really Germans, brought in by John Law, settlers of the "Cote des Allemands." French immigration continued, however, at least up to 1763, when Louisiana was ceded to Spain. It has been said that the southern or Gascon French contributed more than the Norman stock, to the Louisiana colony.

Spanish domination left very little effect on the racial composition of the Territory. The relatively few Spanish officials tended to marry into the French Creole families and be absorbed by them. A small Spanish contribution was made by immigrants from the Canary Islands about 1778.

In 1765 the Acadian-French, forcibly removed from Canada by the English, settled in the Territory and have been a steadily prolific people. A large contingent of Creole families (people of French and Spanish descent born in the colonies) came in from the West Indies during revolutionary uprisings there, 1803-1810.

From the time of the Louisiana Purchase in 1803, there was a steady infiltration of early American stock of British Isles extraction into the Gulf Coast region, and it continued up to the time of the Civil War, together with (during the first few decades of the nineteenth century) some German and Irish immigration. The extent of this "American invasion" is not fully appreciated until one tries to find a real Louisiana Creole to-day.

After the Civil War, the paralysis of Southern industry prevented any further extensive migratory movement in this direction, up to fairly recent times. The present population, then, is rather far removed, in time, from the original European sources.

RACIAL ORIGIN OF THE STUDENTS

The statements made by the students regarding the nationality of origin of their families give no hint of how recently their ancestors came to America and to the South. We have made some attempt during the last two years to get information on this point, and have obtained statements from 440 students chosen as nearly as possible at random,

regarding the birthplaces of their parents and grandparents. About 42 per cent of these students recorded all four grandparents as native to the southern states; about 19 per cent of them recorded four American-born grandparents, one or more of the quartette being non-southern: making 61 per cent "Old American." About 9 per cent had all grandparents foreign-born, and 30 per cent had one, two, or three foreign-born grandparents. If we classify the 1,760 grandparents themselves, it appears that about 65 per cent of them were born in southern states, about 14 per cent in northern states, and about 21 per cent were foreign-born. Of the 372 foreign-born grandparents, 115 came from Germany and 45 from France. It is quite evident that there is a high percentage of Old American stock in this college population, and it should be borne in mind that this limited survey is concerned with students of the last two years only, during which time there have been many more non-local students than in earlier days; while the material from which our study of measurements is taken extends back twenty-two years (1909 to 1930, inclusive).

Due to the mixed ancestry of many of the subjects, the classification of them presented some difficulty. We finally arrived at the divisions indicated in Table 1, which gives the number of subjects in each division. Some of the groups require comment. In that marked "British Isles, Miscellaneous," there are included any combination of English, Irish, Scotch and Welsh, or any one or more of these except English alone. A great many indicated a "Scotch-Irish" ancestry, but because of the carelessness with which this term is used we did not make a separate group of them. The Jewish students were differentiated from others by their statement of religious affiliation, on the college matriculation blank. They were then further differentiated by their statements of national origin on the physical education department records. The "Spanish-Italian" grouping is an attempt to segregate those who had an appreciable degree of South European blood. Almost all of them are mixtures with French, German or North European. The "American" group is of doubtful significance, since it is to be presumed that those who indicated their ancestry thus did not understand what was required, or knew of no foreign ancestry. As the group turned out to have by far the greatest average stature of any, it was retained. The group marked "Unclassified" consists of those who cannot be placed in any of the other divisions. In general they are mixtures

of North and Central European stock, such as English-German-French, etc.

It is evident from a glance at the relative numbers, that the blood of people from the British Isles is predominant in this college population. About 29 per cent of the subjects give only such ancestry, and it enters largely into the "British Isles-French" and "Unclassified" groups also, which make up respectively 17.5 per cent and 17 per cent of the total number. Those of French ancestry alone on both sides comprise only about 4 per cent, in spite of the fact that the great majority of our students are residents of Louisiana. It must be pointed out, however, that if the subjects had been chosen from the Catholic colleges there would have been a higher percentage of the old French stock.

The results of the study of these groups, as regards stature, weight and body build, are brought together in Table 1 and in the bar diagram Figure 1. The statures were distributed into half-inch classes, the weights into five-pound classes, and the individual weight-height ^{2} indices into classes of one English unit, before the means were calculated.

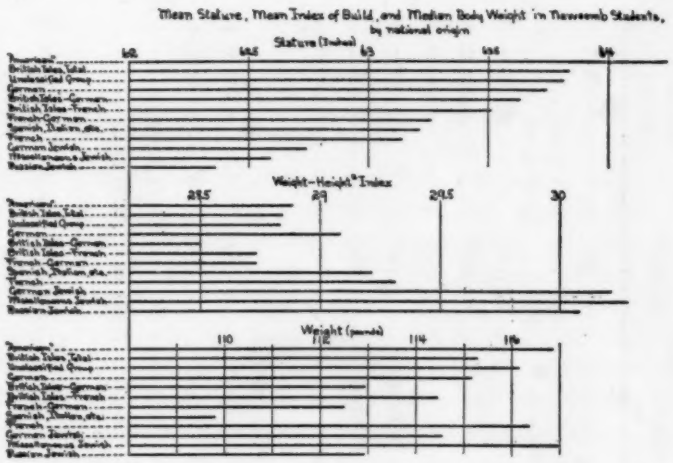
Mean Stature, Mean Index of Build, and Median Body Weight in Nineteenth Students, by national origin
Stature (Inches)
Weight-Height² Index
Weight (pounds)

FIG. 1.

STATURE

The statures of the students of North and Central European origin, excluding the French and British-French, compare favorably with the stature of northern American college women and "Old Americans."

TABLE 1

NATIONAL ORIGIN OF FAMILY	NUMBER	HEIGHT (MEAN)	WEIGHT (MEAN)	WEIGHT (MEDIAN)	INDEX OF BUILD (MEAN)
"American"	150	64.24 ± .12	118.94 ± .88	116.87	28.89 ± .20
English, both parents	268	63.85 ± .09	119.42 ± .76	115.53	29.29 ± .16
British Isles, Miscellaneous	851	63.82 ± .05	116.75 ± .36	114.74	28.71 ± .08
British Isles, Total	1,119	63.83 ± .04	117.39 ± .23	115.27	28.84 ± .07
Unclassified Group	658	63.81 ± .05	117.38 ± .42	116.16	28.83 ± .09
German, both parents	85	63.74 ± .16	117.91 ± 1.19	115.17	29.09 ± .25
British Isles—German Mixtures	256	63.63 ± .09	115.43 ± .73	112.94	28.50 ± .16
British Isles—French Mixtures	682	63.51 ± .05	115.69 ± .37	114.44	28.73 ± .08
French-German Mixtures	103	63.26 ± .12	114.88 ± .94	112.50	28.73 ± .21
Spanish or Italian, not less than ¼, usually mixed with other nationalities	180	63.21 ± .11	116.53 ± .80	109.80	29.21 ± .18
French, both parents	151	63.14 ± .11	116.84 ± .85	116.35	29.31 ± .19
German Jews	109	62.74 ± .15	118.32 ± 1.23	114.53	30.21 ± .29
Miscellaneous Jews	353	62.59 ± .07	118.49 ± .59	116.99	30.28 ± .14
Russian Jews	55	62.36 ± .19	116.68 ± 1.60	112.91	30.08 ± .46
Jews, Total	517	62.59 ± .06	118.36 ± .51	115.96	30.24 ± .12
Total	3,901	63.54	117.05	115.19	29.02

For example, the total mean stature of 15,703 female students of Vassar, Wellesley, Stanford, Minnesota, Smith and Oberlin is 63.78 inches (from data compiled in Table 3, Gould, '30a). The Old American women of Hrdlička ('25) average 63.71 inches. Selecting from our material the British, British-German, German, unclassified European and "American" groups, the total mean height is 63.83 inches. The groups which bring the total average of all the Newcomb students down to 63.54 are the Jewish, the French and the South European mixtures.

Figure 2 shows the distribution curves of stature for some of the larger groups. The differences in average height are accompanied by different positions of the frequency curves, and by different modes, indicating that the mean differences are significant.

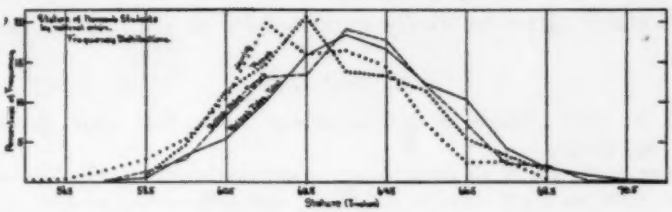

FIG. 2

BODY BUILD

The weights of all the groups are low, compared with northern college women, who average generally over 120 pounds (cf. Gould, '30a). The weight-height ^{2} indices are consequently also low, most of the averages being below 29. It is only the presence of the Spanish-Italian, French and Jewish groups which brings the total average up to 29. Contrast this with the total mean index of build of 6,541 female students from McGill, Wisconsin, Smith, Stanford, Minnesota, Vassar and Wellesley, which is 29.78. It is safe to say that there are some environmental influences which promote slenderness in the Gulf Coast region, in all racial stocks (cf. Davenport and Love, '21).

By comparison of the bar-diagrams of height and body build in Figure 1, it appears that the groups of lowest stature are also the stoutest. Is this because of characteristic stockiness of the South European, French and Jewish groups, or because shortness itself goes with stockiness? To test this, I assembled all the non-Jewish students of low stature, 61 inches or under, and calculating the mean index of build,

found it to be 29.00, practically the same as for all subjects, but slightly greater than for all non-Jewish subjects (28.84). Selecting again the non-Jewish students of high stature, 66 inches or over, I found a mean index of build of only 28.18. It appears that some of the national groups of low stature are stouter than would be expected for their height, but that extreme tallness is itself associated with slenderness.

CONCLUSIONS

A study of stature, weight and body build in female college students of the Gulf Coast states shows that there are mean differences in physique due to diverse national origins which persist even after several generations of American life and intermarriage of the various stocks. It shows also that there is a characteristic slenderness of build in these subjects which cannot be wholly accounted for on the basis of ancestry.

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ANOMALOUS HUMAN SEX-LINKED INHERITANCE OF COLOR-BLINDNESS IN RELATION TO ATTACHED SEX CHROMOSOMES*

BY JOHN W. GOWEN

Color-blindness, inability to distinguish red from green, has been described as due to a recessive sex-linked factor. The evidence for this conclusion is based on the many authentic pedigrees which have been collected since the original description of this condition by Priestly in 1777. A pedigree due to Cunier [1] is of quite a different sort, since in contrast to the mother to son type of sex-linked inheritance the color-blindness descends directly from mother to daughter. The interest of this pedigree lies in the fact that color-blindness, a character whose genetics is as well understood as almost any human character, does not follow its ordinary mode of sex-linked inheritance, but does furnish an illustration dating back a hundred years, of a recently described type of sex-linked inheritance,[2] the attached X type. Before taking up this explanation it is well to examine the previous suggestions to explain this inheritance. Gates [3] has suggested two hypotheses to account for it.

This again appears to be because the character is behaving as a dominant instead of a recessive. The condition is transmitted from mother to daughter directly in the female line—that is, it is sex-linked or, better, female-sex-linked. It may be spoken of as matrilineal in descent. The appearance of the character in heterozygous females shows it to be a dominant. We may think of such females as having one affected and one normal X chromosome. This would be transmitted to half the sons, none of whom are affected, presumably because the presence of the Y chromosome in some way neutralises its activity.[4] Half the sons should then be

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[1] Cunier, F. 1839. Observation curieuse d'une achromatopsie hereditaire depuis cinq generations. *Annales d'Oculistique*, i, pp. 285-288, 1838.

[2] Morgan, L. V. 1922. Non-criss-cross inheritance in Drosophila melanogaster. *Biol. Bul.*, 42: 267-274.

[3] Gates, R. Ruggles. 1931. Heredity in Man. pp. 84-85, *New York* (Macmillan).

[4] There is some evidence that in man and in fishes, unlike insects, the Y chromosome plays an active rôle in inheritance.

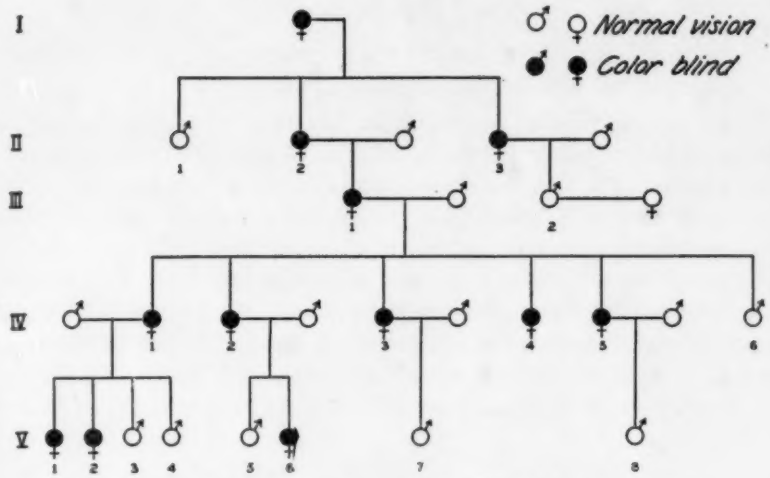

FIG. 1. PEDIGREE OF COLOR BLIND INHERITANCE CITED BY CUNIER

capable of transmitting colour-blindness to all their daughters. Unfortunately the records of the family furnish no data regarding the male lines of descent in this family. But it seems likely that if such transmission had occurred it would have been noticed, because the facts are evidently carefully recorded. It is possible, therefore, that transmission through the males may fail, just as it appears to fail to be inherited through two successive generations in the female line in the night-blind family studied by Newman (1913). Another possible explanation would be that only sons who have received a normal X chromosome are viable. This should lead to a deficiency of males in the offspring.

Castle[5] adopts the hypothesis that the color-blindness gene has become dominant and lethal to males, or that a new gene of this type has appeared.

Apparently in this case the character is behaving as a dominant, though borne as normally in an X-chromosome. If so, we should expect it to be inherited by half the sons. But none of the five sons reported in the pedigree is indicated as affected, which may be due to chance, or more probably to a lethal effect of this allelomorph, when its influence is not offset by a normal X-chromosome, as in the affected females. The sex ratio shown in the pedigree 11 females : 5 males corresponds exactly with the 2:1 ratio expected when a sex-linked lethal is involved.

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[5] Castle, W. E. 1930. Genetics and Eugenics. A text-book for students of biology and a reference book for animal and plant breeders. Fourth edition, p. 362. *Cambridge* (Harvard University Press).

THE ATTACHED SEX CHROMOSOME HYPOTHESIS

The case can be interpreted by still another scheme of inheritance, the hypothesis of attached sex chromosomes.

The human female is thought to have two sex chromosomes, her female character being due to the balance of sex chromosome and autosomal genes. The two sex chromosomes are ordinarily independent, and their free assortment leads to the sex-linked inheritance of color-blindness ordinarily observed. If the sex chromosomes become attached—a phenomenon well known in Drosophila but not hitherto observed in man—the type of inheritance would be altered, since the individuals with attached sex chromosomes are always females. Under these circumstances a male gets his sex chromosome from his father and not from his mother as he ordinarily would. A female gets both of her sex

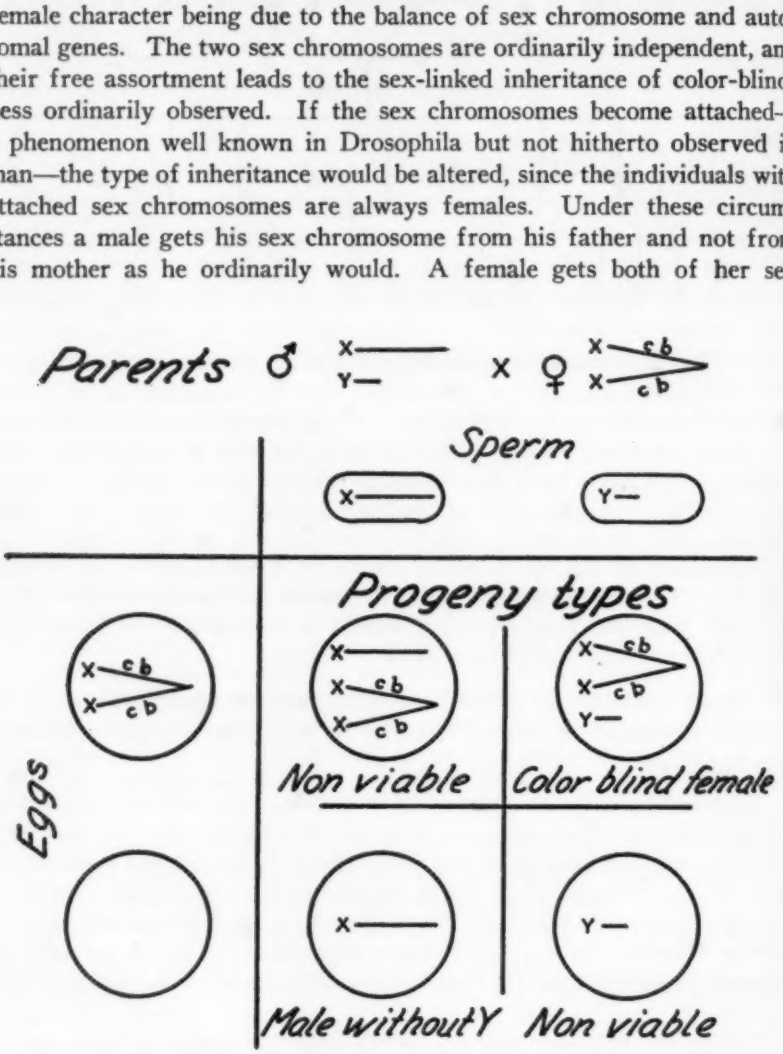


FIG. 2. SEX-LINKED INHERITANCE OF ATTACHED X-TYPE AT INITIAL ATTACHMENT OF THE SEX CHROMOSOMES

chromosomes from her mother instead of one from her mother and another from her father.

The diagramatic representation of such inheritance is shown below, only the sex chromosomes being indicated. It is assumed that attachment originally took place in a female homozygous for color-blindness, cb.

It will be observed that the sons are unaffected and that the daughters are all color blind. Further breeding of these daughters to unaffected males would result in the same types of progeny, except that sons in later generations would receive Y chromosomes from their mothers.

Again, daughters are all color-blind and sons are all normal. This mode of inheritance fits the pedigree of Cunier cited above perfectly.

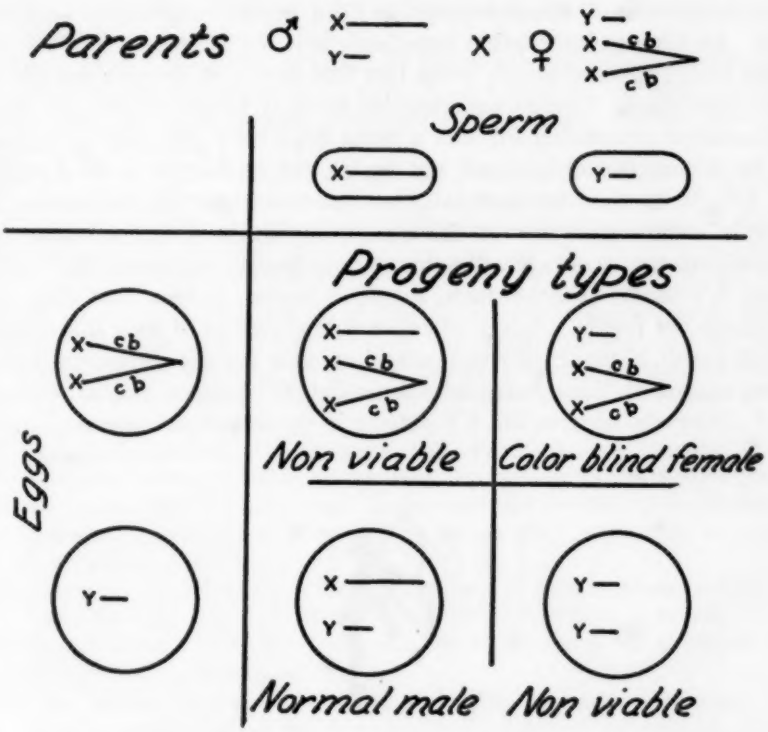
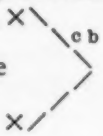
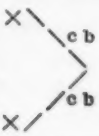


FIG. 3. SEX-LINKED INHERITANCE OF THE ATTACHED X-TYPE WITH Y PRESENT AS IT IS OBSERVED IN DROSOPHILA

It is not surprising that such cases are rare. Attachment of X chromosomes rarely takes place in Drosophila. The original attachment in this case need not have taken place in a female homozygous for color-blindness. Crossing over occurs readily between attached X chromosomes so that if the original female were 

some of her daughters would be 

While the numbers in this pedigree are small they support the attached sex-chromosome hypothesis better than they do the hypothesis of change of dominance with lethal effect. By the attached sex chromosome hypothesis *all* females should be color blind; the pedigree supports this. By the dominant lethal hypothesis half the females should be not color blind; opposed to this is the fact that out of 11 females not one is not color blind. Castle's comment on the 2:1 nature of the sex ratio is somewhat misleading for with a lethal hypothesis the color blind class in the female should be equal, not double, the number of normal males.

Finally another implication of sex chromosome non-disjunction in man is interesting in view of the controversy on the Y chromosome. If non-disjunction occurs it will automatically lead to occasional XO males from XY stock. Furthermore, should it happen in man that these individuals are fertile, a fairly widespread distribution of such individuals might result, a condition which would account for the difference in the observations of Winiwarter who finds the XO pattern and of Painter and others who observe the XY pattern in the sex chromosomes of man.

RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Department of Biology, School of Hygiene and Public Health, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

I. EVOLUTION AND GENETICS

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